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Development of a Standard for the Health Hazard Assessment of Mechanical Shock and Repeated Impact in Army Vehicles

Phase 1

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New tactical ground vehicles developed by the U.S. Army are lower in weight and capable of higher speeds than their predecessors. This combination produces repetitive mechanical shocks that are transmitted to the soldier primarily through the seating system. Under certain operating conditions, this exposure poses health and safety threats to the crew as well as performance degradation due to fatigue. The Army Surgeon General urgently required the Medical Research and Materiel Command to develop exposure standards for repetitive impacts that are relevant to the environment of soldiers operating modern tactical vehicles.

A five-phase research study was designed to develop a standard for the health hazard assessment of mechanical shock and repeated impact in Army vehicles. Phase 1 reviewed aproximately 1,200 papers and documents from relevant scientific, medical, and military literature. The areas of focus were: epidemiology; subjective response; physiology; biochemistry; muscle activity; biomechanics; biodynamic response and modeling; field

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studies; current standards; and signal processing. An annotated bibliography was generated and the existing literature was summarized.

Evidence exists that long-term exposure to vibration accelerates onset of spine disorders, and possibly adversely affect the gastro-intestinal and cardiovascular systems. Few human studies have investigated physiological and biochemical responses to repeated impact, and none have investigated recovery. Distinct differences were noted in the mechanism and outcome of acute and chronic injury to the spine caused by exposure to impact and vibration. Few biodynamic models were designed to predict chronic health problems; however, important contributions have been made to understanding tissue stresses. International Organization for Standardization (ISO) guidelines for human response to whole-body vibration do not adequately account for exposure to repeated impacts. Vertical acceleration and crest-factors in military vehicles can exceed the exposure limit of ISO (2631, 1982) in minutes. Signal processing techniques (e.g., shock spectrum, peak processing and autoregression) identified in other applications will be used in Phase 2 of this study to characterize motion data containing repeated impacts from military vehicles.

This study reviewed, analyzed, and summarized state-of-the-art knowledge of the health hazards associated with whole-body vibration and repeated impact. The review indicated potential approaches to development of a health hazard assessment based on physiological, biochemical, and biodynamic responses. The literature did not support using current vibration exposure standards for motion environments with high magnitude mechanical shocks and repeated impacts. Most existing biodynamic models were not designed to predict chronic health problems. However, certain biodynamic models, which range from single degree of freedom to three-dimensional and discrete parameter models, may have direct relevance to the development of a health hazard assessment index.

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Introduction

The objectives of the project "Development of a Standard for the Health Hazard Assessment of Mechanical Shock and Repeated Impact in Army Vehicles" are:

- to review, analyze and summarize state-of-the-art knowledge of the health hazards associated with whole-body vibration and repeated impact

- to characterize the repeated impact environment of tactical ground vehicles (TGVs) through analysis of recorded acceleration signatures, and to develop methods for realistic simulation of the TGV acceleration environment

- to identify those biomechanical, physiological and pathological human responses which are essential for the prediction of injury risk, and to measure the responses of volunteer subjects under simulated conditions of tactical ground vehicle vibration

- to develop and validate a dose-effect model that will serve to predict the risk of injury to the soldier when exposed to the repeated impact environment of tactical ground vehicles

This report fulfills the requirements of phase one of the project's scope of work and the first objective. The goals of phase one were: to define the potential pathological effects of whole-body repetitive impact in the tactical ground vehicle operating environment; to identify parameters which characterize that environment and to identify measures of biomechanical, physiological and pathological response indicators which should be monitored and which are useful for the prediction of injury risk.

New tactical ground vehicles developed by the U.S. Army are light in weight and are capable of high speeds. This combination results in high levels of impact being transmitted to the soldier. Under certain operating conditions, this exposure poses health and safety risks to the crew and and will cause performance degradation due to fatigue. Current standards for analyzing human response to vibration do not adequately account for the health effects of repeated impact. There is an urgent requirement to develop exposure standards for repetitive whole-body impacts which are relevant to the environment of soldiers operating modern tactical vehicles and weapon systems.

Literature searches have been conducted of a wide range of scientific, medical and military databases. Where possible and practical, foreign articles were obtained and translated. Foreign articles were not obtained if their data were as presented in english sources, such as Dupuis and Zerlett's (1986)

book. Contacts were established with scientists currently conducting research on human response to vibration for up-to-date data and information. Copies of doctoral theses and institutional reports were obtained. Proceedings were obtained from past conferences, such as Advisory Group for Aerospace Research and Development (AGARD) and the United Kingdom Informal Group Meeting on Human Response to Vibration.

The literature was divided into several topic areas, including:

- health effects of vibration and impact from epidemiological research
- subjective response to vibration and repeated impact
- physiological effects of vibration and repeated impact
- biochemical effects of vibration and repeated impact
- muscle response to vibration and repeated impact
- biodynamic response, biomechanics and biodynamic models
- standards and guidelines for shock and vibration
- vibration data collected in the field
- signal processing

Scientists and engineers on the project team reviewed and analyzed the literature relevant to their particular area of expertise. Appendix A lists the primary team members and the areas of literature they reviewed. In each topic area, team members benefited from contributions from other team members. Each topic area was reviewed individually, and conclusions are provided at the end of each section. Where relevant, tables have been constructed to display information from various researchers and facilitate comparison of results. The final section presents overall conclusions and a discussion of the relevance of the literature to development of a standard for the health hazard assessment (HHA) of mechanical shock and repeated impact.

Health Effects of Vibration: Epidemiological Research

Introduction

Epidemiology is the study of the nature and cause of diseases. Knowledge of the health effects of exposure to vibration has largely been determined by health studies of populations exposed to vehicle vibration. Health effects have been classified as either acute or chronic (Griffin, 1990). Acute effects exhibit their maximum response almost instantly and are therefore associated with discrete events. For example, spinal fracture as a result of a single impact from seat ejection is an acute injury. Chronic effects develop over time and are usually associated with cumulative action (Griffin, 1990). Studies to determine chronic effects are largely based on retrospective or cross-sectional surveys (Griffin, 1990). Many of the studies focus on subjective symptoms of health problems, such as backache. Other studies investigate objective findings of disease through clinical or radiological procedures.

Different approaches to epidemiological studies are taken by the Eastern and Western cultures. The East takes a holistic, ecological approach and has characterized a range of symptoms to support the concept of a vibration-induced illness of the whole-body (Japan and U.S.S.R.) (Dupuis and Zerlett, 1986). This approach has not generally been accepted by Western medicine, which tends to approach epidemiology using a cause-effect model (Griffin, 1990). The result is that Eastern studies tend to conduct medical examinations, while the Western studies determine health from medical records. This has resulted in the Western approach focussing on clinically recognizable problems, rather than allowing for the study of biochemical or physiological changes that may precede clinical disease or injury (Dupuis and Zerlett, 1986).

Ideally, if causation is to be unarguably shown between vibration exposure and chronic health problems, extensive epidemiological studies must be conducted on large populations of workers exposed to whole-body vibration. There should be clinical as well as radiological findings, precise occupational histories, vibration measures and typical daily doses, as well as the number of years of exposure. The group examined should be re-examined with the same methods several years after exposure to vibration to measure any changes in health (Dupuis and Zerlett, 1986). A control group must also be measured that is comparable to the exposed group in every respect except for the vibration exposure. Confounding variables due to the occupation and lifestyle of the control group must also be considered. Unfortunately, as several authors have noted, there are few studies of this type (Hulshof and van Zanten, 1987; Dupuis and Zerlett, 1986). Hulshof and van Zanten (1987) set up criteria for evaluating epidemiological

papers and none of the papers they reviewed satisfied their criteria of detailed exposure history, defined medical procedures and adequate study design.

Most of the disorders described in vibration studies are not specific to vibration, but occur generally in the population. They can be associated with or aggravated by other ergonomic or environmental problems (Dupuis and Zerlett, 1986). Studies are also hampered by the problem of workers leaving vibration-exposed occupations with advancing age or onset of disease. Despite the fact that few epidemiological studies satisfy the criteria for determining cause-effect relationships, many studies have resulted in similar findings about the possible chronic health effects (Dupuis and Zerlett, 1986; Hulshof and van Zanten, 1987; Seidel and Heide, 1986), and that inference of causal relationships is valid. The most common health problems are back pain and disorders (such as damage of the intervertebral disc, and degeneration of the spinal vertebrae). To a lesser extent there are also reports of gastro-intestinal disorders, abdominal pain, increased urinary frequency, prostatitis, hemorrhoids, hypertension and cardiac disorders. Health problems will be reviewed in categories of back pain (derived from subjective symptom diagnosis), radiological or clinical back disorders (from objective findings), and other health problems.

Heavy and Miscellaneous Equipment Operators: Back Pain

Studies determining the percentage of operators with back pain have typically been poorly conducted. Few have utilized control groups, few report statistics or details of experimental design and only a couple have measured the vibration exposure of the operators. One of the earliest reports of backache, termed "tractor-back", was by Paulson in 1949. In his rural practice he reported that of 23 tractor drivers, 43.5% had low back pain due to tractor driving since symptoms subside with cessation of operation and increase in heavy seasons or when the ground is rough and hard (Paulson, 1949).

Studies reporting back pain among heavy equipment operators and miscellaneous vehicle operators are summarized in Appendix B, Table B-1; back pain among tractor drivers is in Appendix B, Table B-2; and for truck, bus and car drivers is found in Appendix B, Table B-3. In studies with no control group the percentage of operators with pain ranges from 30-92%. Tractor operators reporting back pain were 30%, 41% and 61% in three studies, all without control groups (Rosegger and Rosegger, 1960; Zimmermann, 1966; Kohl, 1975). Among large populations of container tractor drivers (540) and truck drivers (633), approximately 40% reported pain (Konda et al. 1985; Backman, 1983). Higher levels were found among log stacker operators (70%) and log truck drivers (92%) (Wilcox et al. 1988). This study did measure the vibration exposure and reported that the

International Organization for Standardization (ISO) fatigue decreased proficiency (FDP) boundary was exceeded in all vehicles, and the exposure limit (EL) in some (average vertical acceleration of 0.73 $m.s^{-2)}$. Unfortunately, the numbers of operators were small and no control group was used. Among 600 subway train operators exposed to vertical vibration ranging from 0.3 to 0.7 $m.s^{-2}$, 73% reported pain (Johanning et al. 1991). Flamenco dancers are exposed to repeated impacts through their dancing and roughly one-third reported back pain (Bejjani et al. 1988).

One study was found that discussed "tank-back" and "jeep-back" as common occurrences after the 1939-45 war (Beevis and Forshaw, 1985). Back pain was investigated among trainees at Combat Arms School. Drivers in M113 Armoured Personnel Carriers (pool drivers) had the highest levels of back pain (89%) and these vehicles had highest vibration measurements (average vertical acceleration of 1.1 $m.s^{-2}$ and crest-factors of 13). Pain for another group of drivers was 46%. Although the second group operated the same vehicle, they did so for far fewer hours per day. Pain for Centurion drivers was reported for 55% and the vibration level was considerably lower (average vertical vibration of 0.1 $m.s^{-2}$ and crest-factors of 6). Unfortunately again the numbers of operators were small (less than 30), but a relationship between pain and vibration dose was found.

In six studies that compared back pain in vibration-exposed operators with a control group with no exposure, all found the pain was significantly higher in the exposed group (Boshuizen, Bongers, and Hulshof, 1990; Dupuis and Zerlett, 1987; Riihimaki et al. 1988; Rehm and Wieth, 1984; Burdorf and Zondervan, 1990; Burton and Sandover, 1987). In a questionnaire response from 577 tractor operators, there was a 10% difference in pain between vibration exposed and non-exposed workers (Boshuizen, Bongers, and Hulshof, 1990). A vibration dose was calculated and the prevalence of pain increased with increasing dose (except for severe problems). Pain was most related to increasing duration of exposure. Among 149 operators of earth moving machines and a control group of 215, back pain was 70% and 54% respectively (Dupuis and Zerlett, 1987). Machine operators (longshoremen and earthmovers) were compared with dynamic physical workers (carpenters) and sedentary workers (office) for frequency of low back pain (Riihimaki et al. 1988). Allowing for other risk factors the relative risk was 1.3 contrasting machine operators with both office workers and carpenters, but carpenters had no excess risk compared with office workers. In a large questionnaire study of coal mine workers, 91.9% of heavy engine operators, 87% of truck/car operators and 84.6% of retired truck operators reported pain, compared with 70% of control workers (Rehm and Wieth, 1984). In a recent study of low back pain among crane operators with matched control workers, the odds ratio for pain was 3 to 6 for the vibration exposed operators (60% versus 26.7% in controls)(Burdorf and Zondervan, 1990). Race car drivers are a group exposed to high vibration and shock (average of 2.0

$m.s^{-2}$). Burton & Sandover (1987) found significant differences in their reports of back and neck pain compared with a control group. Less pain was reported in the same group the following year when changes in racing car suspension resulted in a reduction in transmitted shock and vibration.

In some studies in which back injuries were reported, it is not known if the injury was diagnosed from clinical and radiological methods, or from absence from work due to pain. These studies are summarized in Appendix B, Table B-4. Injuries were reported for underground load-haul-dump (LHD) operators compared with three control groups (other underground workers, underground supervisors and above-ground office workers). The incidence rate of back injuries was similar between underground workers (performing heavy materials handling) and LHD operators, however, the relative risk compared with underground supervision was 2.8 and with office workers was 5.0 (Desrosiers, Morrison, and Brubaker, 1988). In a series of studies by Boshuizen et al. (1990) among crane and tractor operators, a significant increase in long-term disability (defined as greater than 28 days) was found for crane operators compared with controls. One study looked at long-term (>28 days) absence from work among crane operators and found absence due to intervertebral (IV) disc disorders lasted for longer periods of time and there were a greater number of disease pensions for compared with controls (Bongers et al. 1988). There was no overall difference in sick leave between tractor operators and controls, however tractor operators had a higher incidence of first-time sick leave. The incidence of lost-time back disability (due to inter-vertebral disc disorders) was higher for tractor and other heavy equipment operators than for controls (Boshuizen, Hulshof, and Bongers, 1990). Hannunkari, Jarvinen, and Partanen (1978) found disability to railway engineers similar to trainmen, but significantly greater than for clerks.

Heavy Equipment Operators: Back Disorders

Radiological back disorders in heavy equipment and miscellaneous vehicle operators are shown in Appendix B, Table B-5; back disorders in tractor drivers are shown in Appendix B, Table B-6; and back disorders in truck, bus and car drivers are shown in Appendix B, Table B-12. One paper frequently cited is a questionnaire survey of 1474 orthopaedic surgeons in the U.S. (Fishbein and Salter, 1950). The authors named some 45 disorders that the respondents thought could be related to driving. A link was made between driving and health problems, yet whole-body vibration exposure was not mentioned in the study. The response rate on the questionnaire was 26.6%.

The largest occupational group subjected to radiological studies of back disorders appears to be tractor operators (Appendix B, Table B-6). Ten studies were found that report the

percentage of radiological back disorders (Rosegger and Rosegger, 1960; Boshuizen, Hulshof, and Bongers, 1990; Dupuis and Christ, 1972; Christ and Dupuis, 1968; Kohl, 1975; Kubic, 1966; Lavault, 1962; Schulze and Polster, 1979; Seidel and Troster, 1970; Zimmermann, 1966). Back disorders varied from 28% to 80%. Many of the European studies were not published in English, but they are reported in a book by Dupuis and Zerlett (1986).

Rosegger & Rosegger's (1960) study of tractor operators remains a landmark for its findings of premature degenerative deformations of the thoracic and lumbar vertebrae, and adolescent kyphosis. Of their 371 operators, 70% were found to have radiological disorders. However, the study did not contain a control group or statistical evaluation of data.

A more recent study of long-time sick leave in tractor operators compared with physical workers found an incidence of 3 injuries per 100 person years for the former, and 2 injuries for the latter (Boshuizen, Hulshof, and Bongers, 1990). The highest incidence was for intervertebral disorders with increasing incidence reported with increasing dose and duration. In view of the fact that the control workers had a high incidence of injuries due to heavy lifting, the findings are important.

Heavy construction equipment operators, such as excavator operators, have also been extensively studied (Appendix B, Table B-5). Radiological disorders were found in 53 to 81% of excavators studied (Kunz and Meyer, 1969; Kohne, Zerlett, and Duntze, 1982; Grigoryan, Druzhinin, and Asadullaev, 1989). A large U.S. National Institute for Occupational Safety and Health (NIOSH) study of heavy equipment operators reported identical percentages of radiological back disorders among vibration exposed (8.4%) compared with controls (8.5%) (Spear et al. 1976). However, the authors conducted follow-up studies to investigate the number of workers leaving vibration jobs and concluded that there may be some disease conditions whose onset is hastened by exposure to vibration even though the overall incidence differs little between exposed and unexposed workers. Dupuis and Zerlett (1987) conducted a large epidemiological study of 352 earth moving operators using interviews and medical examinations. In addition, X-rays were available for 251 machine operators exposed to vibration for at least 10 years. A control group of 215 non-exposed persons was used as a reference. "Lumbar syndrome" was reported in 81% of operators compared with 53% of controls. Lumbar syndrome includes all symptoms which are caused directly or indirectly by degenerative lesions of the lumbar discs, including spondylosis, spondylarthrosis and spondylosteo-chondrosis, in so far as these lesions led to radiologically demonstrable morphological changes in the spinal column and were associated with disc-related complaints (Dupuis and Zerlett, 1987). Since radiologically proven morphological changes of the lumbar spine are a function of age in the general population, Dupuis and Zerlett graphed the incidence with age for the machine operators and controls, as well as for the general population.

The control group and general population curves coincided. In vibration exposed workers, changes are considered to occur earlier and more frequently, demonstrating premature signs of wear in the skeletal system (Dupuis and Zerlett, 1987). A second study also found through evaluation of clinical data and x-rays that degenerative diseases of the spine occur in vibration exposed subjects about 10-12 years earlier than in the average population (Rehm and Wieth, 1984).

Truck drivers' morbidity data was evaluated in another NIOSH study in 1976 (Gruber, 1976). Truck drivers, when compared with air traffic controllers and bus drivers, had higher incidences of premature degenerative deformations of the spinal column, vertobrogenic pain syndromes, and sprains and strains (Gruber, 1976). High levels of radiological problems were reported for truck drivers by Kristen (1981) (83%) and Schmidt (1969) (79.5% compared with 61.1% among controls). Rehm & Wieth (1984) reported 65% of retired truck operators, 77% of truck/car drivers and 80.3% of heavy engine operators had radiological problems compared with 62% of controls. In a small population of drivers (13), 15% reported disc herniations (Wilcox et al. 1988). Data from the studies is summarized in Appendix B, Table B-7.

Bus drivers were studied by Garbe (1981) who reported 50% of drivers had radiological problems, and by Barbaso (1958) who found 44% with problems. Gruber and Zipperman (1992), in another NIOSH study, reported significantly higher incidences of back disorders in 1448 bus drivers compared with 3542 controls. Cavigneaux and Laffont (1969) found 65% of car/taxi drivers with radiological problems.

Only one study was found that looked at tank operators (Beevis and Forshaw, 1985), however their population was small (28) and no control group was used. They found 7.1% with intervertebral disc degeneration. More recently a number of retrospective follow-up epidemiological studies have focussed on crane operators (Boshuizen, Bongers, and Hulshof, 1990). Measures have also been taken of vibration exposure according to ISO measurement techniques. Dose is reported in these studies as year $m^2.s^{-4}$. The studies have concluded that a dose of less than 0.25 is not likely to cause disorders. Levels greater than this are associated with a 10-20% increase in the prevalence of low back pain. Back disorders increased in prevalence above 2.5 years $m^2 \cdot s^{-4}$. Intervertebral (IV) disc disorders were found to increase among crane operators with increasing years of exposure. The relative risk was almost three with a minimum of 5 years of exposure, and almost 5 with 10 years of exposure (Bongers et al. 1988).

In a study of ship personnel, 87% were reported to have radiological back disorders, although no control group was used for comparison (Kersten, 1966). An extensive Japanese study of crew members (487) on high speed ships revealed that after three years, 76% had vertebral deformations (compared with 65% in a

large control group of transport and labour workers) (Kanda et al. 1982). This difference becomes more significant in view of the age distribution; 76% of crew members with deformations were less than 40 years of age, compared with 54% in the control group. More than two-thirds of the deformations were wedge-shaped occurring most often in the first lumbar and 12th thoracic vertebrae. The authors believe the shocks and repeated impacts of high-speed ships (dynamic response index (DRI) permissible exposures less than one hour) cause the deformations at the most vulnerable bend in the spinal column (Kanda et al. 1982).

Pilots: Back Pain

Helicopter pilots are different from other equipment operators in many respects. The vibration amplitudes are generally lower than those in heavy equipment, and the dominant frequencies are higher. The control configuration demands continual involvement of both hands and feet to operate. The location of controls often results in asymmetrical and forward slumping postures for extended periods of time. In Shanahan, Mastroianni and Reading's study (1985), pain was transient lasting less than 24 hours for over half the respondents. The pain is described as a dull ache confined to the lower back with a mean onset time of 88 minutes into flight. Often symptoms do not recur until the next flight, and aviators cannot provoke the symptoms in other activities (Shanahan and Mastroianni, 1984). Temporary pain is thought to be related to an increased incidence of chronic back pain, disorders and disability (Bowden, 1985).

The various studies summarizing pilot back pain are shown in Appendix B, Table B-8. Once again, most of the studies did not use a control group, but simply reported the percentage of pilots with pain. These percentages ranged from 15 to 100 (Singh, 1983; Silosberg, 1962; Fitzgerald and Crotty, 1972; Froom et al. 1986; Jones, 1975; Fischer et al. 1980; Froom et al. 1984; Froom et al. 1987; Shanahan, Mastroianni, and Reading, 1985; Bongers et al. 1990; Boshuizen, Bongers, and Hulshof, 1990; Metges, Flageat, and Mouchon, 1986; Burmeister and Thoma, 1992). Four studies did report significant differences between back pain in pilots and that for controls (Froom et al. 1986; Bongers et al. 1990; Boshuizen, Bongers, and Hulshof, 1990; Fitzgerald and Crotty, 1972). Froom (1986) found back pain higher in helicopter pilots than in transport and fighter pilots, but no outside control group was used. Interestingly, chronic pain was higher in the fighter pilots and the authors attributed this to stresses caused by aircraft ejection. Bongers et al. (1990) calculated vibration doses and found transient pain to be related primarily to the average hours of flying per day, while chronic pain was most related to the total hours of flight (dose). Significant increases in pain were found after 2000 hours or a dose of 400 hours $m^2.s^{-4}$. The authors suggested that the transient pain develops into chronic pain for pilots (Bongers et al. 1990).

Metget, Flageat and Mouchon (1986) found the onset of pain increased between 1400 and 2400 hours total exposure, or after 2 to 3 hours of flight (or 3 to 4.5 hours per day, or 30 to 45 hours in one month).

Some authors mention the possibility of repeated shocks or air turbulance as a cause of back pain (Poirier et al. 1991; Bowden, 1987; Rance and Chappelow, 1974). In the latter study, turbulance was associated with a 50% increase in reports of discomfort. One author reported that accelerations and frequencies common in helicopters can cause microtraumas, although no evidence was given for this (Poirier et al. 1991).

Many of the studies discussing back pain among helicopter pilots suggest the cause is more likely posture, seating design and poor ergonomics in the cockpit, as opposed to vibration (Braithwaite and Vyrnwy-Jones, 1985; Bowden, 1984; Bowden, 1987; Froom et al. 1987; Reader, 1985; Shanahan, 1984; Poirier et al. 1991). Some of the studies do not even mention vibration as a possibility. Reader (1985) found aircrew twice as likely to suffer back pain as ground crew, and pilots more than other aircrew. Froom et al. (1987) did a cross-over study where pilots flew alternately in the front and rear positions. There was an increased prevalence of back pain in the front (pilot's) seat, the onset of pain was quicker and the intensity greater. Both of these studies concluded that posture is the important factor in back pain for pilots. When a seat pad was constructed with added lumbar support, back pain was reported to be diminished for 50% and resolved for 32% of subjects (Reader, 1985).

The classic study investigating whether vibration or posture was the predominant factor relating to back pain among pilots was conducted by Shanahan in a laboratory simulation (1984). A mock-up of a UH-1H seat and control configuration was mounted to a multi-axis vibration simulator. Simulated vibration was similar to "cruise" conditions. Subjects were tested on the apparatus for 120 minutes and in another session received no vibration. There was no statistical difference between sessions in time to onset of pain or intensity of pain. It was concluded that posture is the main etiological factor in back pain among pilots (Shanahan, 1984).

Pilots: back disorders

Clinical or radiological back disorders have also been investigated among air transport operators (Fischer et al. 1980; Froom et al. 1984; Froom et al. 1986; Bongers et al. 1990; Metges, Flageat, and Mouchon, 1986; Froom, Margaliot, and Gross, 1985) (Appendix B, Table B-9). Disorders vary from 0.9% reporting spondylolisthesis in one study, to two studies where 80% were found to have radiological disorders (Fischer et al. 1980).

Significant differences in back disorders were reported by Fischer et al. (1980) and Froom et al. (1986). Helicopter pilots were found to have four-times higher incidences of spondylolisthesis (SLL) than cadets and transport pilots (Froom et al. 1984). Fighter pilots with sciatica had narrowed L5-S1 spaces compared with transport pilots used as controls (45% vs. 3.1%) (Froom, Margaliot, and Gross, 1985). It was concluded that fighter pilots had accelerated degeneration likely due to the g-forces encountered in manoevres. In 1987, Froom et al. reported that pilots with SLL all continued to fly, but they may have been at risk of progression of slippage during ejection. Two studies were found that concluded there was no difference in radiological disorders, even though they found significant differences in pain and lumbago (Bongers et al. 1990; Metges, Flageat, and Mouchon, 1986). A study comparing the spines of parachutists with controls found statistically significant changes in the thoracic and cervical region, but not in the lumbar region (Mustajoki, Nummi, and Meurman, 1978). Stresses to parachutists are primarily due to impact with the ground.

Other Health Problems

Appendix B, Table B-10 details studies investigating health problems other than back pain and disorders among equipment operators and pilots and Appendix B, Table B-11 shows health problems in truck, bus and car drivers. Many of the studies simply document the incidence of health problems among vibration-exposed workers. Three provided comparison with a control group (Gruber, 1976; Corbridge and Griffin, 1986; Corlett and Rose, 1985), and another three German studies, presented by Dupuis & Zerlett (1986), reported significant differences for gastro-intestinal problems compared with a control group (Garbe, 1981; Kohne, Zerlett, and Duntze, 1982; Kohl, 1975). The large NIOSH studies of the 1970s (Milby and Spear, 1974; Gruber and Ziperman, 1992) were the broadest attempt to investigate health effects among vibration-exposed populations. They utilized large numbers of operators and proper control groups. Unfortunately, data were obtained from insurance records, disability claims, questionnaires to physicians and periodic medical examinations that were already coded into disease categories. Variability arises due to misclassification of disease, under-reporting by workers and lack of standardization among physicians.

Milby and Spear (1974) investigated heavy equipment operators and found an elevated risk in 3 of the 30 disease claims: ischemic heart disease, obesity of non-endocrine origin and certain musculo-skeletal disorders (such as displacement of the intervertebral disc). The trend, however, did not increase with increasing exposure. A follow-up study was conducted to determine if workers exposed to vibration were selecting themselves out of jobs due to disability (Spear et al. 1976). The results lacked significance to conclude a definitive selection process, however,

the authors concluded the unusual trends indicate whole-body vibration (WBV) hastens the onset of certain diseases, but does not increase the overall incidence (Spear et al. 1976).

A NIOSH study of bus drivers, sex matched and age adjusted to comparison groups, found significant differences in digestive, circulatory and musculo-skeletal systems, specifically gastric neurosis, varicose veins, hemorrhoids, acute respiratory infections, peptic ulcers, appendicitis, urological surgery, inguinal hernia and displacement of IV disc (Gruber and Ziperman, 1992). In investigations of interstate truck drivers compared with air traffic controllers, significantly higher incidences of hypertension, cardiac diseases, sprains and strains, appendicitis, stomach troubles and hemorrhoids were found in the vibration exposed group (Gruber, 1976). Although these studies did report significant findings, the authors were not able to conclude that vibration was responsible since drivers are also exposed to poor dietary habits, noise and other environmental factors, stress and prolonged poor posture.

Other studies, with less rigorous methods and no control groups, also report health problems in vibration-exposed workers concentrated in the digestive, cardiac, renal/urino/genital, respiratory and hearing loss areas (Guidotti and Cottle, 1987; Rehm and Wieth, 1984; Hannunkari, Jarvinen, and Partanen, 1978; Backman, 1983; Paulson, 1949; Kunz and Meyer, 1969; Christ and Dupuis, 1968 Kubic, 1966; Sakuma, 1980; Rosegger and Rosegger, 1960; Seidel and Troster, 1970; Schmidt, 1969); the latter five studies reported by Dupuis and Zerlett (1986). The German investigations of tractor operators all report gastro-intestinal problems as the health problem second to back disorders. Rosegger and Rosegger (1960) report pathological findings in the stomachs of 76.1% of tractor drivers. Acute findings included gastroenteroptosis (dropped stomach), hypersecretion, gastritis, hypersecretory gastritis and combinations with a dropped stomach. Chronic findings included bulbus duodeni (condition after the course of a duodenal ulcer is over). An increase in stomach disorders with increasing years of exposure was found, and this is supported by Christ and Dupuis (1968).

Relationship between whole-body vibration and health disorders

It has been concluded by several authors that whole-body vibration is one of the potential etiologic agents in development of some medical conditions in vehicle operators. Unfortunately the epidemiological literature available to date does not allow accurate differentiation of contributing factors, sufficient to ascribe a dose-effect relationship. This is largely due to poor documentation of the dose of vibration, the many environmental stressors (such as noise, psychological stress, and prolonged sitting) acting in combination, and the difficulties in obtaining

an ideal control group (Gruber and Ziperman, 1992). A control group performing the same tasks as the exposed group, except for WBV, is difficult, if not impossible to find. Also, many other factors predispose or contribute to the health problems associated with WBV. There are no known health effects specific to WBV. Different body systems seem to react to WBV somewhat independently of one another. Signs and symptoms of diseases attributable to WBV are common in control groups not exposed to WBV (such as back pain and gastro-intestinal disorders) (Griffin, 1982). From 60-80% of the normal population is expected to suffer low back pain at some time (Wilder, Pope, and Frymoyer, 1982). It has been suggested that fit workers may select themselves into vibration-exposed jobs more than unfit workers (Griffin, 1982). Studies are confounded by the fact that many people choose leisure activities (such as motor cycling and ski-doo riding) that expose them to WBV. Prolonged inactivity, or conversely, heavy materials handling in some non-vibration jobs, lead to similar incidences of some of the health disorders. Studies are further confounded because workers change jobs: from vibration exposed to non-exposed, possibly due to injury or disease, and between exposed and non-exposed jobs when healthy. Workers with vibration-induced injuries could therefore be within control groups during the sampling period.

Few epidemiological studies have taken account of other physical activities of drivers (Troup, 1985) or considered the rate of increase in acceleration (impacts) as a possible etiological factor for health disorders (Troup, 1985; Troup, 1988). A good study would include a job analysis for postural and dynamic spinal loads and a more appropriate measure of exposure would be developed (Troup, 1988). It is also important to account for age when constructing dose based on years of exposure, since many of the health disorders increase in the normal population with aging (Griffin, 1982). Other factors include age at the beginning of the occupation, spinal system integrity, living habits and an individual's ability to withstand vibration (Dupuis and Zerlett, 1986). Troup (1985) feels there is little evidence for a causal relationship between on-road driving and back pain. He does admit, however, that the contributory role of shock from off-road surfaces remains undetermined (Troup, 1985).

Seidel and Heide (1986) conducted one of the largest reviews of the epidemiology literature related to WBV. They selected 78 papers for their core review. Vibration exposure was characterized in only 27 of these papers, in another 22 they could estimate it, and it was unknown in 29. None was measured according to ISO 2631 criteria and almost none included measurements in the x or y direction, crest-factors, dominant frequencies, or shocks. Daily exposure time was mentioned in half the studies and 35% used statistical analyses. The authors went on to estimate the exposure according to the ISO exposure limit (EL). They reported that 35% exceeded the EL, 15% were near it and 35% remained uncertain. From their review of the health problems in these papers, they concluded that WBV at or near the

EL causes an increased health risk to the musculo-skeletal system and peripheral nervous system, with a lower probability of risk for the digestive system peripheral veins, female reproductive organs and vestibular system. The health risk seemed also to increase with increasing intensity or duration, with hints of non-linearity (Seidel and Heide, 1986).

Possible etiologies of health disorders due to WBV

Despite the problems with epidemiological studies, it has been argued by some authors that there is sufficient evidence to conclude that long-term exposure to WBV can be harmful to the spine and, possibly, other systems of the body (Hulshof and van Zanten, 1987; Dupuis and Zerlett, 1986; Seidel and Heide, 1986). There is some acceptance that vibration, rather than causing specific pathologies, accelerates the onset of currently recognizable syndromes (Sandover, 1981; Troup, 1988; Spear, Keller, and Milby, 1976). In 1980 the International Labour Organization recognized the occupational origin of diseases of the spinal column as a result of vibration exposure, especially in drivers of power-driven machinery and tractors (Dupuis and Zerlett, 1986). Troup (1988) explains two possible mechanisms. In the first, disturbance of proprioception caused by vibration might increase susceptability to microtrauma of the vertebra. In the second, the static and reactive muscular activity induced by vibration while driving might lower one's resistance to impact. During impact the muscles may not have sufficient time to respond in a protective manner.

The most convincing hypotheses offered as etiologies for back disorders have been discussed by Dupuis and Zerlett (1986) and Sandover (1981). One hypothesis is based on disturbances in capillary blood flow (similar to vibration induced white finger). This hypothesis is further supported by evidence that WBV disturbs both peripheral and central blood flow (See physiology section). It has also been suggested by Sandover (1981) that dynamic compression loading of the intervertebral joint leads to fatigue-induced microfractures at the endplate or of subchondral trabeculae. Callus is formed during the healing that may lead to reduced area for nutrient diffusion. In addition, the end plates may be disturbed and have altered diffusion characteristics. This process would accelerate the normal degeneration in the nucleus and annular fibres (See Biomechanics: Nutrition and Fatigue Failure). A mechanical hypothesis is that dynamic shear, bending and rotational loading of the joint leads to fatigue and breakdown of annular lamellae, acceleration of annular degeneration and sometimes nuclear prolapse.

As with back problems, it is impossible to ascribe stomach problems solely to vibration since other occupational, environmental and individual factors contribute to the same effects. Drivers of heavy equipment are typically exposed to

multiple stresses, work shifts, poor diets, psychological stress, prolonged seated posture and lack of exercise. However, many authors feel WBV is a factor in the etiology of stomach disorders. Resonant frequencies of the stomach often are similar to the vehicle vibration and mechanical strain is imposed. Rosegger & Rosegger (1960) believe long term exposure can alter the reaction of the vegetative nervous system causing a change in tone of smooth musculature, with a secondary effect of activity of the stomach-intestinal canal.

No papers were found that postulated etiologies for renal, urinary, genital, cardiac, and respiratory disorders related to WBV. It is plausable that disturbances may be related to mechanical movement of the organs, especially in the resonance range. Alternatively, the reduced blood flow and nutritional hypothesis of back pain may contribute to problems in other organs of the body as well (Knapp, 1974; Hansson and Holm, 1991).

Conclusions

Epidemiological studies were reviewed to determine the health effects of chronic exposure to vibration and repeated impact. Many studies have been conducted of heavy equipment operators from industries such as agriculture, construction, mining, forestry and the military, and from aircraft pilots, (predominately of helicopters). Many studies focus on subjective symptoms of health problems, such as backache. Other studies investigate objective findings of disease, such as back disorders (intervertebral disc herniation and spondylolisthesis), as diagnosed through clinical or radiological findings.

Most of the disorders associated with vibration are not specific to vibration, but occur generally in the population. They may be associated with other ergonomic or environmental problems. Few studies have used appropriate control groups of workers not exposed to vibration. Vibration levels and years of exposure are often not reported in the studies, and some fail to adequately define the type of equipment the population is operating. Some studies suffer from poor study design and lack of statistics.

Despite the fact that few studies satisfy the criteria for determining a cause-effect relationship, and a dose-response cannot be determined for vibration and health effects, some conclusions can be drawn. Sufficient evidence is available to conclude that long-term exposure to vibration can be harmful to the spine and possibly other systems of the body (gastro-intestinal and cardio-respiratory). There is no single disorder linked to vibration or impact. Instead, there is some acceptance that vibration, rather than causing specific pathologies, accelerates the onset of currently recognizable syndromes. Although some studies include exposure to vibration and repeated

impact, there have been few studies where repeated impact is a variable under consideration. Several hypotheses have been developed to explain the etiology of back disorders. One hypothesis suggests vibration alters nutrition to the disc. A second suggests dynamic loading of the intervertebral joints causes fatigue damage to the annulus of the intervertebral discs.

Subjective Response to Mechanical Vibration and Shock

Introduction

The vast majority of research about human response to vibration has been based on peoples' subjective responses to the motion. This focus has been important historically for defining tolerance in certain situations. For example, it was one method used in studying the effects of the shock of seat ejection from military aircraft. It was also important to automotive and vehicle designers attempting to quantify and improve vehicle ride. These data were first applied to the development of standards describing peoples' response to vibration in the 1930s. Forty years later, when the International Organization for Standardization drafted its "Guidelines for Human Response to Vibration" in 1974, much of the data used were obtained from these subjective studies conducted in the laboratory and the field. Today, many studies continue to be published in this area mostly to equate various conditions such as rotational with translational vibration, or intermittent shocks with sinusoidal vibration. These studies contribute important knowledge about the various factors governing human response to vibration and shock.

Limitations of Subjective Data

Early experiments suffer from differences and inadequacies in experimental technique, such as too few subjects, poor equipment, lack of control over the length of exposure, variable instructions and failure to document the variability of results (Oborne, 1976). Early studies also used semantic labels such as "extremely uncomfortable" which resulted in large variance due to differing definitions of comfort (McCullough, 1974) and to difficulties relating comfort to the sensation experienced because of a vibration (Oborne, Boarer, and Heath, 1981). Asking subjects to match sensations from different vibration is a conceptually difficult task (Fothergill and Griffin, 1977; Howarth, 1986). Oborne stated that subjective comfort and attempts to draw equal comfort contours have not significantly increased the knowledge of how people react to whole-body vibration because of such limitations (Oborne, 1976).

Humans do not have a single receptor organ for vibration, as we do for hearing. The effects of vibration on various parts of the body, therefore, differ depending upon characteristics of the exposure. Higher frequency vibration (10 Hz) tends to affect the head while lower frequencies (5 Hz) affect the back (Dupuis and Zerlett, 1986). Differences arise between individuals because some may be more sensitive to head sensations than back sensations.

Most subjective studies have been conducted in a laboratory under sinusoidal vertical axis motion. There is evidence that rotational axes and vibration of the feet and of the back influence the perception of comfort (Parsons, Griffin, and Whitham, 1982; Parsons and Griffin, 1982; Griffin, 1986; Oborne, 1976; Ashley and Rao, 1974). Posture also affects vibration sensation. There can be an individual difference of 20:1 in transmission of vibration due to postural adjustments (Griffin, 1975). Postural factors include supination angle, standing versus sitting, erect versus slouched, coupling to the seat, hanging feet, existence of backrest, restraint, harness, and foot vibration (Griffin, Whitham, and Parsons, 1982; Harrah and Shoenberger, 1981; Griffin, 1975). Other variables affecting subjective response to vibration include subject size, height and weight, male versus female, instructions given, past experience with vibration, expectation and attitude, personality, fitness, presentation of stimuli (method of adjustment, reference-test order, uncontrolled stimuli and durations), interaction with rotation, carry-over effects with repeated measures, rigid versus compliant seating and environmental factors such as noise, and climate (Griffin, Whitham, and Parsons, 1982; Griffin, Parsons, and Whitham, 1982; Oborne and Boarer, 1982; Dupuis, Hartung, and Louda, 1972; Griffin, 1990).

Subjective responses are typically used to develop contours that describe the average response of a group. Inherently, there will be some types and levels of vibration considered unacceptable to some subjects whose response is widely different from the average response (Oborne and Boarer, 1982). Dupuis (1986) reports intra-individual variation (within an individual) as 20% and inter-individual variation (between individuals) as 42%. Despite good control over many variables thought to affect subjective response, more than 60% of the variance was found to be, as yet, unexplained (Oborne, Boarer, and Heath, 1981)

Why then has so much laboratory research been conducted to uncover peoples' subjective response to vibration and shock? Studies conducted in a laboratory with systematic procedures can more successfully control the wide range of extraneous factors already discussed that affect human response to vibration in a field experiment. It is easier in a laboratory study to systematically vary one factor at a time and measure the subjective effects. A large number of different types of vibration can be evaluated which is difficult to arrange in field experiments (Griffin, 1990). Controlled laboratory experiments using subjective measures are quick to conduct, cost-effective, repeatable (especially if matching techniques are used) and a large number of subjects can readily be utilized.

In practical terms, there is a use for subjective measurements. Such experiments help to define and determine what types of vibration exposure will be acceptable to the majority of individuals. This has been useful to equipment manufacturers, especially for automobile manufacturers. It is important to

adjust technology to both the objective and subjective requirements of man (Dupuis and Zerlett, 1986). It is difficult, however, to link results of these subjective studies to current objective efforts to describe the health hazard effects of vibration and shock. It has been suggested that people exposed to high mechanical vibration can often trace health problems back to the vibration stress (Dupuis and Zerlett, 1986). It is also thought that at the limits of subjective tolerance, pain in certain parts of the body is a warning of impending damage should the vibration and shock continue. However, it may be argued that lower levels of vibration that may contribute to chronic health problems, or an earlier onset of health problems, may not seem subjectively uncomfortable to the persons exposed at the time of exposure. In such cases, the body's feelings about the vibration may bear no relationship to the potential for eventual damage. Despite the limitations, these studies do provide an important adjunct to the objective health findings since one's health is a function of both physical and psychological well-being.

Definition of Comfort

Paradoxically, comfort is most often defined as a relative absence of discomfort. It is a feeling that is individual and non-specific. Reports of discomfort in vehicle ride have been related to numerous variables, including number of motor and perceptual tasks being undertaken (Rebiffe, 1980), seat design (Andersson, 1980), height and weight of operator and support for operation of controls and other devices (Grandjean, 1980), safety protection, (Habsburg and Middendorf, 1980), and noise and visual stimuli (Griffin, 1990). It is difficult, if not impossible, to calibrate comfort either objectively or subjectively. There is no assurance that a subject who reports vibration-caused discomfort in a field or laboratory experiment is eliminating all other potential sources of discomfort (Habsburg and Middendorf, 1980)

Techniques for measuring comfort

Semantic Labels

Early laboratory and field studies sought to determine comfort levels by presenting the subject with a vibration stimulus and asking for a description using semantic labels such as "perceptible", "mildly annoying", "alarming", "uncomfortable". Many of these types of experiments were designed to answer specific questions, for example, what level of sinusoidal vibration in a simulated aircraft situation is "mildly annoying"? (McCullough, 1974).

Like their laboratory counterparts, field studies have been conducted where subjects were asked to ascribe a semantic label to a vehicle ride, or where a correlation was sought between subjective reports and objectively measured vibration. Unfortunately, many of these studies suffer from poor reporting of methodology (Oborne, 1976). Surprisingly, despite a lack of standardized methods, curves reported from the different studies are more similar than those from the laboratory data (Oborne, 1976).

Psychophysical Methods

Psychophysical studies have been conducted to determine the shape of equal contour vibration curves, with little emphasis on ratings of comfort level. Most often, a subject is presented with a reference stimulus and asked to equate a second stimulus to the reference, or to multiply or fractionate the reference by some predetermined value (such as double or half) (Oborne, 1976).

The results of psychophysical methods produce far less variability in responses than semantic labels, especially within an individual, and the curves tend to remain fairly stable over time (Oborne, 1978). This is partially because one vibration sensation is being compared directly with another. When using semantic labels, a stimulus is compared to a label, then another stimulus is compared to the same label, and the two stimuli are then assumed to be equal (Oborne and Humphreys, 1976). Since different frequencies of vibration excite different parts of the body to different degrees, intensity matching in essence requires a subject to judge sensation in one region of the body to be equivalent to a very different sensation in another part of the body (Fothergill and Griffin, 1977). Because of this difficulty, many researchers have insisted on highly trained subjects or have reused the same subjects in various experiments (Dupuis, Hartung, and Louda, 1972; Miwa, 1968).

A more recent development in psychophysical methods is known as the floating reference technique (Donati et al. 1983). Pairs of stimuli (test and reference) are presented with frequency differences which are no more than one octave. This technique has been found to produce minimal variability over large numbers of subjects (Donati et al. 1983).

Attempts have also been made to equate subjective sensation to intensity of a stimulus (using Steven's power law) (Jones, 1975; Hiramatsu and Griffin, 1984; Oborne, 1976). In this procedure, the subjective sensation is a function of a constant multiplied by the stimulus magnitude to some exponential level. Subjective intensity therefore is proportional to a power function of the stimulus intensity. For example, if an exponent of 2 is selected, than the sensation varies with the square of the stimulus. It has been assumed from this that the power law, once derived, could be

extended in each direction to describe families of curves (McCullough, 1974).

Subjective response to sinusoidal vertical (a_z) vibration

Maximum discomfort to sinusoidal vertical vibration in individual experiments was found at 4 Hz by Meister et al. (1984), at 2, 4 and 8 Hz by Metz et al. (Metz et al. 1986) and at 5 Hz according to Dupuis (Dupuis, Hartung, and Louda, 1972). Dupuis (Dupuis, Hartung, and Louda, 1972) and Rao and Jones (Rao and Jones, 1978) also noted a second area of sensitivity between 0.5 and 2.0 Hz, and the latter reported increased sensitivity above 10 Hz compared with the ISO 2631 (1978) curves.

Equal sensation contours have been produced by various researchers and were reviewed by Oborne (Oborne, 1976). The contours were found to be of two distinct shapes. The first shape indicates maximum sensitivity in the range from 6 to 15 Hz, with a slow reduction in sensitivity below 6 Hz and above 15 Hz. The second shape suggests a smaller range of maximum sensitivity, between 4 and 6 Hz, with a rapid decrease above 6 Hz and below 4 Hz.

Exponents determined using intensity matching and the Steven's power law relationship yielded a variety of results, depending upon methods used, from 0.93 to 1.75 (Jones, 1975; Hiramatsu and Griffin, 1984; Fothergill and Griffin, 1977). Several authors have found the contours to change in shape with increasing intensity (Oborne, Boarer, and Heath, 1981; Oborne, Heath, and Boarer, 1981; Shoenberger, 1979; Rao and Jones, 1978), or with differing instructions (Oborne and Boarer, 1982). Some have found no difference between the responses of males and females (Jones, 1975; Parsons and Griffin, 1988). However, some have found small sex differences, with females being more sensitive above 3.15 Hz (Corbridge and Griffin, 1986).

In a unique study, subjects were asked to indicate on pictures of a seated figure where they felt discomfort at various frequencies and intensities (Whitham and Griffin, 1978). Similar locations were found even with a four-fold increase in vibration level (Whitham and Griffin, 1978). Discomfort was found in the lower abdomen when exposed to 2 Hz, moving up the body with 4 to 8 Hz vibration, and at the head with 16 Hz. At 32 Hz, responses were divided between the head and lower abdomen, while at 64 Hz, they were mostly at the ischial tuberosities (Whitham and Griffin, 1978). Similar results were reported earlier by Parks (1962) using vibration magnitudes at levels ranging up to "alarming". When a backrest was provided, the areas of discomfort were altered. With the backrest angle fairly upright the vibration-induced sensations are mainly reported to be in the upper back, neck and sacrum (Harrah and Shoenberger, 1981). The authors did not present any explanation for the differences.

Subjective response to sinusoidal horizontal (a_x and a_y) vibration

The human body is less sensitive to horizontal vibration (a_x and a_y) than to vibration in the vertical (a_z) direction. Although fairly similar, some studies have reported slightly more sensitivity to y-axis vibration than x-axis (Rao and Jones, 1978). Most researchers have found the frequency of maximum sensitivity to be approximately 2 Hz and that sensitivity decreases with increasing frequency above 2 Hz (Corbridge and Griffin, 1986; Whitham and Griffin, 1978; Rao and Jones, 1978; Miwa, 1969). However, Donati et al. (1983) found maximum sensitivity was 3-4 Hz when using a floating reference technique.

Feelings of discomfort for vibration along both horizontal axes are predominantly in the lower abdomen and buttocks irrespective of frequency or direction (Whitham and Griffin, 1978). There do not seem to be changes in the shape of the contours as a function of vibration amplitude (Griffin, Whitham, and Parsons, 1982). The response among males and females is similar, although there have been differences reported due to size with larger subjects more sensitive than smaller subjects (Griffin, Whitham, and Parsons, 1982).

Subjective response to random vibration

Many studies have compared the subjective response of subjects to sinusoidal vibration (usually in the vertical axis) with that of random, multi-frequency vibration. The purpose of many of these comparisons was to determine which of two suggested analysis methods in the ISO 2631 (1978) Standard better predicts discomfort; the dominant single one-third octave frequency rating procedure, or the frequency-weighted root sums of squares method. Results differ in magnitude, but almost all studies conclude that humans are more sensitive to random motion than sinusoidal, and that the frequency weighting method correlates better with subjective response to vibration (Griffin and Whitham, 1977; Corbridge and Griffin, 1986; Dupuis, Hartung, and Louda, 1972; Shoenberger, 1976; Shoenberger, 1978; Metz et al. 1986; Donati et al. 1983; Fothergill and Griffin, 1977; Miwa, 1968). There is some evidence that the frequency weighting method also correlates better correlated to biological measures, e.g., electromyography of muscles, than the one-third octave rating method (Metz et al. 1986). Two studies reported no significant difference in sensitivity of subjects to sinusoidal vibration, compared with random, although they still found the frequency-weighted method of ISO a better predictor (Meister et al. 1984; Mistrot et al. 1984).

Subjective response to multiple directions of vibration

As with multi-frequency vibration, much of the investigation on the effect of multiple directions has been to determine how best to predict discomfort with vibration from more than one direction. When subjects were exposed to vertical and horizontal vibration simultaneously and frequency was varied, the frequency of the horizontal vibration was more important than vertical in predicting discomfort (Kirby, Coates, and Mikulka, 1975). Dual axis motion (y and z) was rated more severely than single components (either y or z), and discomfort was not found to be greatly influenced by the phase between the two single-axis components (Shoenberger, 1987; Griffin and Whitham, 1977).

Most studies have found the root sum of squares method the best predictor of multi-axis discomfort (Fairley and Griffin, 1988; Griffin and Whitham, 1977; Mistrot, Donati, and Galmiche, 1990). Interactions between some frequencies, however, have been reported; there is a tendency for some higher fore-aft frequencies combined with lower vertical frequencies to cause less discomfort than expected by rms, but some higher vertical frequencies combined with lower fore-aft frequencies caused greater discomfort than expected (Fairley and Griffin, 1988). Mistrot, Roley and Donati (1990) found that the linear sum of components overestimated discomfort, and the root mean quad (rmq) underestimated it. Unfortunately, they used a small range of vibration exposures and might have found different results with a larger range.

Effect of duration on discomfort

The time dependency of vibration is one of the most controversial topics in the literature. The ISO Standards (1978) offer limits of exposure for periods from 1 minute to 24 hours. Generally, three types of experiments have been conducted: field surveys where passengers are asked to record their comfort levels at various times during a trip; laboratory experiments where subjects rated comfort over a short duration; and laboratory experiments where subjects ability to perform simple tasks is analyzed over different exposure times (Clarke, 1979).

Clarke (1979) reports that only two long-duration studies provided any evidence for increasing discomfort with increasing exposure duration, however; no variability was presented in the data (range or standard deviation) and no non-vibration control situation was included. Increasing discomfort could therefore have been due to poor seating or tiredness, irrespective of vibration exposure. Two duration studies which used exposures of one hour and 36-minutes respectively, actually found an increase in comfort over the duration of exposure (Clevenson, Dempsey, and Leatherwood, 1978; Griffin and Whitham, 1976). They attributed

the results to adaptation and highly motivated subjects. The studies that have found a relationship between duration and discomfort, such as Hiramatsu and Griffin (1984), measured extremely short exposure durations (2-50 seconds).

Two studies in particular took a comprehensive look at the effects of long duration and repeated exposures of vibration (Guignard, Landrum, and Reardon, 1976; Schulte-Wintrop and Knoche, 1978). Neither found support for an increase in discomfort with increasing duration of exposure. However, the later study showed that fatigue among subjects increased with increasing duration. Breaks in exposure did not seem to justify higher permissible doses (Schulte-Wintrop and Knoche, 1978).

Howarth (1986), and Griffin and Whitham (1980) criticize the time dependency with rms and ISO Standards, but go on to suggest that the rmq relationship has more validity with time. The same criticisms can be applied to the rmq as to the rms method of the ISO Standards; evidence is based on a few studies and short duration exposures.

Subjective response to rotational vibration

Two research laboratories, in particular, have carried out over the past 13 years multi-phase studies evaluating the equivalence of various combinations of rotational vibration for the purposes of developing equivalence contours (Griffin, Parsons, and Whitham, 1982; Parsons and Griffin, 1982; Shoenberger, 1978; Shoenberger, 1980; Shoenberger, 1984; Shoenberger, 1985; Shoenberger, 1986; Shoenberger, 1987). Rotational motion about the x-axis, y-axis and z-axis is referred to as roll, pitch and yaw, respectively.

Roll motion causes greater discomfort at the same level and frequency than pitch motion (Parsons and Griffin, 1978; Parsons and Griffin, 1978). However, tests were conducted with no backrest. When a backrest and restraint were used, subjects reported more sensitivity to pitch vibration (Shoenberger, 1980). The least sensitive motion is yaw (Parsons and Griffin, 1982). Although there were no reported differences in contours among males and females, if subjects were larger, had longer lower legs or the height of a footrest was increased, less discomfort was reported from pitch. The likely explanation is that there was less contact between the subject's legs and the seat (Parsons and Griffin, 1982; Parsons and Griffin, 1978).

Shoenberger (1979; 1979) found roll axis discomfort increased as a function of frequency and intensity, while Parsons and Griffin (1978) found sensitivity decreased with increasing frequency. Shoenberger (1984; 1986) also conducted a range of studies rotational combined with translational vibration was

matched with a sinusoidal translational vibration (z-axis). The main findings were:

1. For y+roll and for y+yaw motion the vibration was more comfortable as frequency increased.

2. For y-axis compared with y+roll, response can be accounted for by y-axis translational vibration alone.

3. There was a higher response for x+pitch than for x-axis, likely due to input from the seat back.

4. Discomfort increases with distance from the axis of rotation.

Equal intensity contours were plotted by Griffin, Parsons and Whitham (1982) for three axes of rotational motion. It has been suggested that comfort could be predicted by the most severe component in a combined exposure, the rms of the combined axes, or an rms of 12 axes (three translational at seat, three rotational at seat, three translational at backrest and three translational at footrest) summed and the root taken for an equivalent total rms (Parsons and Griffin, 1978; Griffin, Parsons, and Whitham, 1982).

Ride comfort models

A valid question concerning all the laboratory comfort studies is whether the data generated will be applicable to actual vehicle rides in the field. Oborne (1978) reviewed contours published from a number of sources and concluded that an intensity level of approximately 0.6 to 0.8 $m.s^{-2}$ borders on uncomfortable. However, this relationship is dependent on situation. Car riding comfort was judged to be acceptable to 90% of the public at a level of 0.5 $m.s^{-2}$ for durations up to four hours (Cooper and Young, 1980). Sometimes passengers will tolerate higher levels of vibration in the field since extraneous stimuli may distract their attention (Oborne, 1978). The upper level of comfort in laboratory studies with inexperienced subjects is approximately 0.25 $m.s^{-2}$, while subjects exposed in a laboratory to simulated field conditions were comfortable with higher levels of vibration (0.6 $m.s^{-2}$) and passengers in the field were comfortable at 0.9 $m.s^{-2}$ (Oborne, 1978).

A great deal of effort has gone into development of various ride comfort models, mostly for automobiles (Pepler et al. 1986; Varterasian, 1982; Healey and Young, 1975; Jacobson, Richards, and Kuhlthau, 1980; Leatherwood, Dempsey, and Clevenson, 1980), but also for aircraft (Stone, 1974), trucks (Mistrot et al. 1987; Corbridge, Griffin, and Levis, 1980), buses (Levis and McKinlay, 1980) and tractors (Monsees et al. 1988). Some of the models have looked at environmental exposures such as noise, pressure and

temperature in addition to vibration to predict passenger comfort (Jacobson, Richards, and Kuhlthau, 1980). Models, such as Leatherwood's (1980), have not been field validated. When vibration measured in the field was simulated in a laboratory, good correlations were reported with the discomfort ratings (Corbridge, Griffin, and Levis, 1980; Stone, 1974).

Many of the field studies attempting to construct ride models report that the best correlation with subjective comfort is the vertical acceleration on the seat (Levis and McKinlay, 1980; Parsons and Griffin, 1980; Pepler et al. 1986; Stone, 1974) and in some cases the vector sum (of x, y, and z-axis vibration) was almost as good as vertical acceleration (Healey and Young, 1975). In other cases transverse acceleration (Pepler et al. 1986) and acceleration of the backrest and of the feet were important as well (Parsons and Griffin, 1980). Two studies also found the rate of change of acceleration, or jerk, important (Pepler et al. 1986; Levis and McKinlay, 1980).

When several methods of characterizing data (rms, rmq, vector sum, etc.) are compared with subjective ratings in the field, weighted rms is usually reported to provide the best correlation (Mistrot et al. 1987; Monsees et al. 1988). Findings in these studies depend upon the levels of vibration, frequencies, crest-factors, and number of characterization methods compared.

Discomfort from shocks and impulsive vibration

There has been increased activity in this area of research during the 1980s, likely associated with use of rmq and development of the vibration dose value (VDV) methodologies which were adopted by the British Standards Institute (B.S. 6841, 1984). Much of the controversy over consideration of shocks or impulsive vibration in a time signal stems from lack of an internationally accepted definition for impulsive vibration (Maeda, 1989). Some researchers believe to give shocks a correct weighting it is important to know the critical rise time (Kjellberg and Wikstrom, 1985; Miwa, 1968)

It has been generally thought that random vibration with peaks is subjectively rated as more severe than random vibration with no peaks, even when both have the same rms (Clarke et al. 1965; Griffin and Whitham, 1980). Greater discomfort has been associated with higher peak levels (Griffin and Whitham, 1980), and with increasing numbers of peaks (Griffin and Whitham, 1977). The repetition rate of the shock (Corbridge, 1982), and the separation time between shocks (Yonekawa, 1979) has been found to be important. When impulses were separated by greater than 0.625 seconds, discomfort increased (Corbridge, 1982).

Other studies have reported that peaks are difficult to discriminate in a vibration signal, and may not make the ride

less comfortable (Duncan and Wegscheid, 1985; Monsees et al. 1988; Sandover and DeKoker, 1991). Higher background acceleration tends to make peak accelerations more acceptable, probably by masking them (Monsees et al. 1988). Sandover and DeKoker (1991) suggested subjective responses may not be a good way to discriminate between signals with varying severity of shocks; however, the signals used in their study had low crest-factors (5-8). Lines, Whyte and Stayner (1987) report a large spread of subjective assessments for a small change in acceleration and were not confident of their data.

A number of studies have attempted to determine which method best characterizes vibration containing repeated impacts (Miwa, 1968; Griffin and Whitham, 1977; Kjellberg and Wikstrom, 1985; Wikstrom, Kjellberg, and Orelius, 1987; Howarth, 1987; Monsees et al. 1988; Griffin and Whitham, 1980; Lines, Whyte, and Stayner, 1987). There is no general consensus among the findings. Shocks were found to be underestimated when using an rms procedure (Miwa, 1968; Kjellberg and Wikstrom, 1985). Griffin and Whitham (1977) found that the exponent was between 2 and 4. In a very complete study, Wikstrom (1987) found little difference between an exponent of 2 and that of 4.

The rmq or VDV were reported to have a better relationship with discomfort when compared with rms and other measures by several investigators (Griffin and Whitham, 1980; Hall, 1987; Hoddinott, 1986; Howarth and Griffin, 1991). However, Griffin and Whitham (1991) stated that if there are only occasional shocks in the exposure, the discomfort will be overestimated by rmq It seems reasonable that a dose measure would be more appropriate than an averaging measure, since an average measure depends to a large extent on the period over which the average is obtained (Howarth and Griffin, 1991). Monsees et al. (1988) and Lines (1987) found that rms was a better predictor of discomfort than rmq and Monsees et al. (1988) used signals with crest-factors up to 28. The statistical procedure used to correlate subjective comfort with the measurement procedure (Pearson's Correlation) in these studies often resulted in very similar correlation coefficients. Therefore, even though a study concludes one method is superior to another, there is usually very little practical difference between measures. One study reported an exponent of unity for different frequencies, durations and directions of motion and concluded that a single frequency weighting and method for incorporating the effect of duration is appropriate at all shock magnitudes (Howarth, 1987).

A very thorough evaluation, worthy of elaboration, involved discomfort ratings during two field studies and used a large number of vibration characterization methods (Wikstrom, Kjellberg, and Orelius, 1987). Methods included various peak value analyses, weighting methods by time-mean analysis and dose analysis (with exponents from 2 to 10), and response analysis based on models and impulse analyses. Mean and dose calculations yielded higher correlations with discomfort ratings than peak,

impulse and response analyses. In one of the field studies, the mean and dose could not be differentiated, while in the other, dose analyses correlated best and the correlation weakened as th exponent increased from 2 to 10. The authors concluded that a measure sensitive to peaks in the signal is less appropriate tha one taking the whole acceleration signal into account. There was no significant difference in correlations between vibration and comfort when using a dose of 2 and of 4.

One possible reason that laboratory studies have found higher correlations with an exponent of 4 is that subjects in a lab cannot anticipate a shock the same way as a vehicle driver can. Predictability and control likely reduce one's subjective assessment of a shock. It has also been pointed out that the weighting method of the ISO 2631 (1978) may not be appropriate for signals with shocks since it may attenuate the important components of impact and result in an underestimation of discomfort (Hall, 1987; Howarth and Griffin, 1991).

The effects of shocks superimposed on a random signal remains a controversy. Most studies report that shocks increase the discomfort, although to a lesser extent if the background vibration is high. The relationship between discomfort and the frequency and magnitude of shocks is uncertain. Some believe an rmq overestimates the addition of shocks to the signal (Griffin and Whitham, 1980; Lines, Whyte, and Stayner, 1987; Monsees et al. 1988) and others that an rms underestimates the discomfort (Griffin and Whitham, 1980; Hall, 1987; Hoddinott, 1986; Howarth and Griffin, 1991). To others, exponents of two and four seem equally associated with discomfort (Wikstrom, Kjellberg, and Orelius, 1987). Variables confounding the study of impacts include the range of signals used in the study; whether the study is conducted in the laboratory where subjects cannot anticipate the shocks, or in the field where they can; and how experienced the subjects are with the motion.

Tolerance to vibration and shock

Tolerance, like comfort, is a relative term that tends to depend upon the environment in which the shock and vibration exposure takes place (Janeway, 1975; Cole, 1978). For example, tolerance in an accident may mean survival, even with injuries, while tolerance in normal transport environments is akin to comfort, and building vibration tolerance is much lower again (Cole, 1978).

Tolerance levels come from three sources: subjective tolerance to various durations of sinusoidal vibration in laboratory experiments; data from accidents such as survivable aircraft escape, automobile impacts, and free falls from lifeboats; and models of the response to vibration and impact. Levels of

tolerance from each of these sources are shown in Appendix B, Table B-12.

Several attempts have been made to define subjective tolerance levels to short-time (less than one minute) sinusoidal vibration (Janeway, 1948; Dieckmann, 1958; Goldman and von Gierke, 1961; Magid, Coermann, and Ziegenruecker, 1960; Parks, 1962; Miwa, 1968). Subjects were either exposed to a range of vibration levels and asked to match the level with a semantic label such as "alarming", or they were exposed to increasing levels of sinusoidal vibration until they stopped the motion at their tolerance limit.

From the laboratory studies of tolerance, there seems to be little agreement on levels or on frequencies that subjects are most sensitive to at the tolerance limit. There is some consensus for a tolerance limit in the vertical axis at 3.3 to 4.0 $m.s^{-2}$ centered about the 3 Hz range (Dieckmann, 1958; Janeway, 1948; Dupuis, Hartung, and Louda, 1972). Work by others resulted in tolerance limits ranging from 6 to 9 $m.s^{-2}$ (9 at 2-3 Hz) (Parks and Snyder, 1961; Gorrill and Snyder, 1957). A third group of studies have found vertical tolerance between 12 and 20 $m.s^{-2}$, depending upon the frequency (Chaney, 1964; Magid, Coermann, and Ziegenruecker, 1960; Forshaw and Ries, 1986; Beaupeurt et al. 1969).

Tolerance limits will vary depending on what exposure duration is used. Levels for 3 minute tolerance vary from 1.8 to 12 $m.s^{-2}$ (Magid, Coermann, and Ziegenruecker, 1960; Miwa, 1968; Forshaw and Ries, 1986). Thirty minute tolerance tests conducted by Dupuis, Hartung and Louda (1972) yielded values of 2 to 4 $m.s^{-2}$, with subjects most sensitive to 5 Hz. However, subjects were most sensitive to 2 Hz during short-time (less than one-minute) tests (Dupuis, Hartung, and Louda, 1972). On the other hand, Magid et al (1960) found most sensitivity at 7-8 Hz during very short-time tolerance tests and at 5-7 Hz during 3 minute tests.

Fewer studies report tolerance levels in the horizontal directions. Miwa (1968) reported three-minute tolerance limits for the horizontal directions at 2.8 $m.s^{-2}$, which is a higher tolerance than he found in the z direction (1.8 $m.s^{-2}$). It has been suggested that we have higher tolerance to x-axis motion, but y and z axes are similar (Temple et al. 1964).

Descriptions of sensations at the limit of tolerance provide useful information about the possible mechanical and physiological response of the body. The magnitude of acceleration tolerance, and to some extent the type of symptoms reported, are strongly influenced by experimental design, support or restraint systems used in the experiments, and frequency (Temple et al. 1964). Generally, at lower frequencies (4-8 Hz) the centrally located organ-tissue systems are more affected, and as frequency is increased, the peripherally located organs become affected to a greater extent (Forshaw and Ries, 1986; Parks, 1962).

Reasons for the various symptoms that have been offered by Forshaw and Ries (1986) are:

Abdominal pain: stretching and deformation of the terminal ileum, cecum, hepatic flexure and transverse colon

Chest pain: stretching of major vessels originating at the base of the heart and mechanical stimulation of diaphragmatic pericardium

Testicular pain: displacements of the spermatic cord and deformation of the testicles

Head symptoms: displacement of facial skin and subcutaneous tissues about underlying bony structures

Dyspnea: alternating displacements of thoraco-abdominal system and pulmonary hemodynamics such as pooling of blood in pulmonary vessels resulting in pulmonary congestion

Anxiety: pain, stimulation of proprioceptive system and respiratory impairment.

It has been reported that the major symptoms in the y-axis are due to pressure in the thorax, while the x-axis symptoms are respiratory and complaints originating from the head (Temple et al. 1964).

Tolerance to impacts, or abrupt accelerations, depends largely on the acceleration time history and therefore, a critical time is often quoted with the tolerance levels. For example, 15 g for 0.2 seconds can often be survived with no injury during aircraft ejection. But 15 g for 2 seconds would render the victim unconscious (Cole, 1978). Apart from the duration of impact, the important factors in sustaining damage include magnitude of force, impacted structure, direction of force (body orientation), distribution of force, age, sex, physical condition and mental condition of subject, tissue properties and secondary inputs (Snyder, 1963; Society of Automotive Engineers, 1986). Tolerance to brief impact is more related to velocity change, while for tolerance to longer impacts, acceleration is important.

Tolerance limits were in a similar range for pilot ejection seats (7 to 25 g, based on a time of 17 to 25 ms) (Ames, Sweeney, and Savely, 1947), survivable aircraft escapes (15 to 22.8 g in vertical and side to side directions and 28 to 46 g in fore-aft) (Brinkley, 1985), lifeboat free fall (9 to 18 g) (Nelson, Hirsch, and Magill, 1988), and NASA-MSC and AMRL experiments (3 to 26 g) (Weis et al. 1964).

Models for predicting tolerance were usually based on spinal injury, such as the dynamic response index (DRI). Discussion of this and other models can be found in the Biodynamic Models Section.

Subjective effects of noise in combination with vibration

Given that noise is often present along with vibration, the influence of this added stressor on the subjective perception of vibration and of the whole environment, is important. However, few studies have assessed this interaction in a systematic manner.

Generally, the presence of noise, in addition to vibration, has produced confounding effects on perception of either stimulus level. Guignard (1974) reviewed the area and found that the interaction of the two stimuli varied from antagonistic, through additive to synergistic although he does not detail what factors contribute to this wide range of responses. Fleming and Griffin (1975) attempted to determine the equivalence of each stimulus by matching levels. However, there were no details given about interactions between noise and vibration. Howarth and Griffin (1990) found that vibration does not affect judgement of noise magnitude but noise does affect judgement of vibration magnitude. A combination of both stimuli produced different interactions depending on the relative levels of each. These findings have wider implications on results from studies where noise has not been controlled and vibration levels have been assessed subjectively. For example, if noise were present at any perceptible level during subjective estimations of vibration levels, the vibration may have been rated more severely than in a study where noise was not present at a significant level.

Conclusions

A review of the literature concerning subjective response to vibration, shocks, and repeated impact has been important for several reasons. Many researchers have attempted to characterize comfortable, or acceptable levels of vibration and shock. The International Standards governing vibration (ISO 2631) are largely based on data from subjective comfort studies. A wide range of conditions and types of vibration have been studied in a controlled fashion in laboratories with large numbers of subjects. Finally, descriptions of sensations at the limit of tolerance provide useful information about the possible mechanical and physiological response of the body.

A great deal has been learned from studies of subjective response to vibration. Frequencies of maximal discomfort have been defined for the three translational and rotational axes. Subjects are usually more sensitive to random vibration than sinusoidal, and to multiple directions of vibration compared with a single direction. The increased discomfort imposed by additional frequencies or directions is best predicted by the root sum of squares method. There seems to be little evidence to support an increasing level of discomfort with increasing

duration of exposure (except for exposures of a few minutes). Of the rotational axes, subjects are most sensitive to pitch and roll vibration, then yaw. The location of the axis of rotation is important in determining subjective comfort of rotational motion. Vibration of the seatback, and to a lesser extent the feet, can be important factors in determining discomfort. Ride comfort models derived from field and laboratory studies have generally concluded that vibration levels between 0.6 and 0.8 $m.s^{-2}$ are on the border of being described as uncomfortable. However, one's definition of comfort depends on the environment.

There have been fewer investigations of subjective response to shock or repeated impact. There is no accepted definition of shocks and impacts. Random vibration with shocks superimposed tends to be more uncomfortable than random vibration alone. A number of studies have attempted to determine which method best characterizes vibration with repeated impacts. Exponents of two and four (rms and rmq) seem equally associated with discomfort. The exponent of best fit depends upon the separation time of the shocks, the range of signals presented to the subjects, background vibration levels, frequencies of the shocks, anticipation of shocks and subjects' experience with the motion.

Data of subjective tolerance to vibration and shock have been derived from subjective comfort studies, accidents such as aircraft escape and free falls from lifeboats, and mathematical models of body response. Generally, at lower frequencies (4 to 8 Hz) the centrally located organ-tissue systems are most affected, and as frequency is increased the peripherally regions become affected to a greater extent. Tolerance to impacts depends largely on the acceleration time history. Models, such as the dynamic response index (DRI) have focussed on spinal injury due to single impact, and been extended to applications of repeated impact.

Results from studies investigating subjective response are extremely variable for a number of reasons. Studies have concluded that subjective response may not be an effective way to discriminate the effects due to repeated impact. It is difficult to link results of these studies to current objective efforts to describe the health hazard effects of vibration and shock. Lower levels of vibration and repeated impact may contribute to chronic health problems, or an earlier onset of problems, but may not seem subjectively uncomfortable to the persons exposed. There is no evidence in the literature to support a time dependency of long-term reduced tolerance and few attempts have been made to investigate the subjective response to repeated impact. Despite the limitations, these studies do provide an important adjunct to objective health findings since health is a function of both physical and psychological well-being.

Physiological Effects of Vibration and Repeated Impact

Vibration and impacts have been shown to have acute effects on a number of different systems in the body. Numerous reviews are available (Guignard, 1972; Guignard, 1985; Guignard, 1974; Weaver, 1979; Barnes, 1987; Ramsay and Beshir, 1981). Although somewhat dated, the review by Guignard (1972) provides an overview of the effects of vibration on different systems of the body. Physiological effects are related to two potential mechanisms: the movement of organs and tissues and a generalised stress response related to intensity and duration of vibration exposure. For the present review, physiological effects have been divided into the following categories:

- General
- Cardiovascular
- Respiratory
- Gastrointestinal
- Combined effects of noise and vibration
- Effect of mental workload

General

General physiology

Many of the responses to whole-body vibration are attributed to stimulation or over-activation of the sympathetic nervous system. This can result in increased concentrations of catecholamines and vasoactive metabolites which in turn cause a generalised stress response, and other specific outcomes as detailed in the Biochemistry Section. An increase in heart rate, cardiac output, respiration rate and oxygen uptake occurs in response to WBV. In some cases, peripheral vasoconstriction (Spaul, Spear, and Greenleaf, 1986; Abu-Lisan, 1979) has also been reported. Other effects can be related to the mechanical deformation of different body parts in response to vibration especially at resonant frequencies.

Pathology

Acute pathological effects of vibration and shock include hemorrhagic damage and degenerative changes in organs and systems. These have included injury to viscera, lung and myocardium (Guignard, 1972), bleeding in the gastro-intestinal

system (Sturges et al. 1974), and occasionally, hemorrhage of kidney and brain (Guignard, 1972). The majority of this work has been conducted using animals (Boorstin, Hayes, and Goldman, 1966) and in some cases, high levels and long duration WBV exposures. The hemodynamics of central and regional blood flow are also shown to be disrupted by the mechanical effects of vibration. Aantaa, Virolainen and Karskela (1977) reported damage to the labyrinth of the inner ear when exposed to angular acceleration. He suggests that the damage is due to either mechanical trauma or circulatory disturbances, or both. This will be discussed in more detail in the Combined effects of noise and vibration section.

Cardiovascular

The acute effect of vibration on the cardiovascular system has been compared by many authors to the effect of moderate exercise (Sharp, Patrick, and Withey, 1974; Hood et al. 1966; Barnes, 1987) and this response is related to the increase in muscular activity needed to maintain posture (Duffner, Hamilton, and Schmitz, 1962). Heart rate and cardiac output increase (Bennett et al. 1978a; Bennett et al. 1978b; Bobick, Gallagher, and Unger, 1988; Bobick et al. 1988; Auffret, Demange, and Vettes, 1974; Barnes, 1987; Hood et al. 1966). Blood pressure may also be affected but this response is more variable. Bobick, Gallagher, and Unger (1988), Bobick et al. (1988) showed an increase in systolic and mean blood pressure when subjects were exposed to simulated vibration from a coal haulage vehicle. Abu-Lisan (1979) showed a decrease in diastolic blood pressure following exposure to vertical vibration (4-8 Hz, 0.183 $m.s^{-2}$ rms) which he related to an increase in peripheral resistance. McCutcheon (1974) disputes the similarity between the cardiovascular response to vibration and exercise. Unlike that observed during exercise, his work with animals (discussed below) does not show a consistent direct dependency between changes in mean aortic blood flow, or heart rate, and that in oxygen consumption. Although vibration can induce muscular contraction in order to adjust posture, the energy cost of such activity is quite low and does not match the measured oxygen consumption. In terms of increased cardiac output, it is possible that mechanical pumping of the heart may be caused by vibration. This may well be more extreme with shocks or higher levels of vibration.

Some authors (Watts, 1972) have shown a decrease in heart rate over the duration of the vibration exposure. This has been attributed to measuring resting heart rate just before the start of the experiment when subjects are apprehensive. Heart rate will usually decrease from an initially raised level as the subjects become accustomed to stress (Hornick and Lefritz, 1966; Holland, 1966; Abu-Lisan, 1979). Abu-Lisan (1979) found a decrease in heart rate and cardiac output which he related to either a concomitant decline in central nervous system arousal

and peripheral sympathetic tone, or the measured increase in sodium and calcium ion levels. He suggested that the increase in these ions would decrease heart rate as the tissues became less easily depolarised. Heart rate is affected by many different variables, both physiological and psychological. There is usually both a large inter- and intra-subject difference in the heart rate results so it is unlikely that heart rate on its own would be a meaningful measure to evaluate the cardiovascular effects of vibration and repeated impact.

McCutcheon and co-workers (Bhattacharya, Knapp, Edwards) have performed a number of animal experiments, measuring blood flow, pressure, and electrocardiogram (ECG) during WBV exposure (Edwards, McCutcheon, and Knapp, 1972; McCutcheon, 1974; Bhattacharya et al. 1975; Bhattacharya et al. 1976; Bhattacharya et al. 1977; Bhattacharya et al. 1979). In their 1972 study, they found that +/- 30 $m.s^{-2}$ at 4 Hz caused dramatic changes in pressure and flow from a two-fold increase to a 90% decrease. This difference in response direction was attributed to resonant effects of the large organs. Flow rate and pressure appear to be dependant on the phase relationship between cardiac and vibration cycles. The operational implications of such a cardiovascular phenomenon are explained by McCutcheon with the following example: if a subject is performing a critical and boring task, and a near constant phase relationship exists between cardiac cycle and the vibration for a period of about 10 to 15 seconds, then cardiac output could be reduced by 90%. This would result in insufficient cerebral blood flow, with the concomitant decrement in performance or loss of control of the vehicle. On the other hand, cardiac output/blood pressure could be increased two-fold, which may also degrade or disrupt performance. Bhattacharya et al. (1975) also determined that vibration could be used as a forcing function to produce and maintain a particular cardiovascular response, which they suggest can be beneficial in some clinical situations.

Circulation

Spaul Spear and Greenleaf (1986) and Abu-Lisan (1979) found that WBV caused peripheral vasoconstriction. The vibration exposures ranged from 1.4 $m.s^{-2}$ rms at 5 Hz to 37 $m.s^{-2}$ rms at 80 Hz in Spaul's study and 0.183 $m.s^{-2}$ rms at 4 to 8 Hz in Abu-Lisan's experiment. If peripheral vasoconstriction does occur, this has implications with respect to thermoregulation and homeostasis of operators. In Spaul's study (1986), core temperature was significantly elevated with vibration. Although severe damage has been seen with subjects exposed to localised vibration, for example persons suffering from Vibration White Finger (VWF), there is little evidence in the epidemiological literature to suggest a greater number of circulatory problems in operators exposed to whole-body vibration. Nevertheless, Lysina and Parliuk (1973) found that female crane operators exposed to 'jerky vibration' (undefined) developed 'angiodystonic' syndrome

(disorder of the circulation system). The long-term health implications of vibration-induced vasoconstriction are not clear.

ECG

Vibration and impacts have been shown to have a number of different effects on the ECG signal. Changes have been seen in the R-R interval (Ullsperger, Seidel, and Menzel, 1986; Harada, Kondo, and Kinura, 1990), heart rate variability (Harstela and Piirainen, 1985; Auffret, Demange, and Vettes, 1974), P-R interval (Abu-Lisan, 1979) and T-wave amplitudes (Roman et al. 1968). Moderate low frequency vibration can change a sinus arrhythmia to a sinus tachycardia. Severe exposures and jolts can produce transient brachycardias (Guignard, 1972), extrasystoles (Guignard, 1972; Roman et al. 1968), and affect sinus respiratory arrhythmia (Roman et al. 1968). The implications of repeatedly inducing these cardiac irregularities are not discussed by the authors.

The effect of stress and fatigue on ECG signals has attracted more attention than the effect of vibration and impacts. However, the ECG analysis techniques described below can be applied when examining the effects of vibration and impact effects. Early work by Sayers (1973), and Luczak and Laurig (1973) indicated that the amplitude peaks in spectral plots of tachogram time series (R-R interval plot as a function of time) provided information on the degree of stress or fatigue to which a person is subjected. Over the course of activity, the amplitude peaks will vary due to fluctuations in the body's circulatory control system. Luczak and Laurig (1973) identified several time domain statistics which are correlated with mental stress. The most significant of these are the mean value of the differences between two successive heart beats.

In addition to simple statistics, various spectral analysis techniques are used to evaluate temporal fluctuations in the tachogram (Sayers, 1973; Luczak and Laurig, 1973; Egelund, 1982; Cerutti et al. 1989; Cerutti et al. 1988). Three of these authors (Luczak and Laurig, 1973; Sayers, 1973; Egelund, 1982) report the use of Fast Fourier Transform (FFT) analysis to calculate the amplitude spectrum of the R-R interval tachogram. In typical plots a major peak is discernible, which is related to blood pressure fluctuations at about 0.05 to 0.15 Hz. Peaks due to the thermal regulation system and respiration are also prominent. Luczak and Laurig (1973) showed that good results can be obtained using FFT provided that suitably long records are available for the required spectral resolution.

Baselli and Cerutti (1985) and Bartolli , Baselli and Cerutti (1985) demonstrated that auto-regressive techniques can be used to evaluate the spectral content of the R-R tachogram. Once the parameters for the auto-regression of the tachogram have been estimated, the spectrum can be determined. An algorithm for this

procedure is reported by Marple (1980). This method of spectral estimation is considered superior to FFT methods when only short time records of the tachogram are available for analysis. However, no papers could be found which compared the two methods.

Heslegrave (1987) looked at changes in respiratory sinus arrhythmia and in the baroreceptor reflex rhythm in response to sleep loss. He concluded that centrally-mediated cardiac reflexes can be used to monitor cortical recovery following sleep loss. The studies reviewed here, show that ECG is sensitive to a variety of stress-induced effects and that these can be elucidated by detailed analysis of the signal.

There is not enough evidence to state equivocally that shocks or vibration have a long-term, detrimental effect on the cardiovascular system. Although some epidemiological evidence has suggested an increase in cardiovascular problems, these are not significant in number or effect No studies, to our knowledge, have examined the effects of multiple shocks on the ECG waveform. The immediate effect of shocks on the cardiac system could be assessed by analysing changes in the temporal components of the signals. One technique, which appears to have potential with respect to looking at the effect of shocks on the systems responsiveness, is the phase plot (Goldberger, Rigney, and West, 1990). Examining the effect of vibration on the spectral components of the signal, for example, respiration and thermoregulation, also shows promise. It is suggested that the variability of these peaks decreases when subjects are exposed to various stresses. There may also be a differential effect on these peaks according to the effect of vibration and shocks on the respiratory and thermoregulatory systems themselves. There are obviously a number of choices for ECG signal analysis both in the frequency and time domains. At this stage, it is not clear which method will be most suitable for examining the effects of shocks on the ECG signal. Although it is relatively easy to obtain a clean ECG signal under continuous vibration, the effect of the shocks, especially, on the measuring system has to be determined, to ensure that the quality of the signal is adequate for the type of analysis that we may undertake.

Respiratory

The effect of WBV on the respiratory system is to increase respiration rate, pulmonary ventilation, and oxygen uptake (Duffner, Hamilton, and Schmitz, 1962; Hoover, 1962; Hood et al. 1966; Cole and Withey, 1977). Most studies extrapolated an increase in oxygen uptake from the increase in pulmonary ventilation. As suggested by several authors (Duffner, Hamilton, and Schmitz, 1962; Guignard, 1972; Hoover, 1962), if vibration causes a mechanical pumping of the lungs mediated through movements of the diaphragm and large organs, then there is likely to be increased deadspace. In this case, gas exchange may not be

complete, and alveolar ventilation would be relatively unchanged. To determine whether alveolar ventilation had been altered, a closed-circuit system with complete gas analysis would be necessary. This method has not been used by the majority of authors which raises questions as to the validity of their extrapolated results The mechanical pumping hypothesis ties in with that of McCutcheon (1974), in that the reported increased oxygen uptake exceeds the metabolic demands of the body, even accounting for the increased muscular activity required to maintain posture.

A number of authors have also observed hyperventilation (Sharp, Patrick, and Withey, 1974; Duffner, Hamilton, and Schmitz, 1962; Hoover, 1962; Page, Cole, and Withey, 1977), especially at low vibration frequencies (1-10 Hz). In some cases, the hyperventilation decreases over the exposure time and, like the effect of heart rate, may be an adaptation to the stress (Duffner, Hamilton, and Schmitz, 1962). A number of other theories have been postulated including:

1. centrally mediated effect due to stimulation of somatic mechanoreceptors, including those in lungs and respiratory passages

2. centrally mediated effect due to general muscle stimulation, not necessarily related to oxygen demand

3. mechanical effect due to pumping of the lungs and airways caused by movement of the diaphragm and large organs in response to the vibration which increases carbon dioxide elimination and depresses the respiratory function during recovery

4. anticipatory or anxiety response to the vibration.

Hoover and Ashe (1962) found that the respiratory response varies depending on the vibration frequency and amplitude. Lower amplitude vibration increases respiration rate whereas tidal volume is increased at the higher amplitude. Although some hyperventilation occurred, not all the data were consistent. Hoover and Ashe suggest that the apparent hyperventilation is due to the mechanical effect or stimulation of the autonomic nervous system (ANS).

Bennett et al. (1978a; 1978b) looked at the effects of simulated vibration and impacts from a military tracked vehicle travelling cross-country. Oxygen uptake, minute ventilation, respiratory rate, tidal volume and tracking error were increased. The magnitude of responses increased with increasing vibration level. As in most experiments, inter-subject variances were large, but in this case, the individual's responses were correlated with their 'locus of control'. Locus of control (Rotter, 1966) is based on the premise that there are two types of people: those who believe that they can influence the

environment around them and those who feel that there is little they can do to control the environment. The former are said to have an internal locus of control and the latter an external locus of control. Subjects with an internal locus of control performed better on the tracking task but at greater physiological cost as indicated by a higher heart rate. Those with external locus of control showed the opposite effect.

In the Bennett and Cole studies (Bennett et al. 1978a; Bennett et al. 1978b; Cole and Withey, 1977) no difference was found in the effects of vibration between random and sinusoidal signals. Manninen (1984; 1985), however, found differences in effect between sinusoidal and random vibration. Most of the effects were not consistent over the experimental conditions used and no explanation was proposed. One finding that does support other studies is the decrease in heart rate caused by sinusoidal vibration and the increase by random vibration; the theory being that monotonous vibration decreases arousal.

The long term effect of increasing respiration rate and a possible increase in oxygen consumption may cause fatigue but there is no indication of more serious health effects. If vibration and impacts causes hyperventilation then performance is likely to be degraded.

Gastro-intestinal

Pathological studies including one by Sturgess et al. (1974) on Rhesus monkeys, have shown gastro-intestinal bleeding when exposed to 130 hours of vibration (1.5 g_z peak at 12 Hz). Effects included blood in the feces and a drop in hematocrit. Much of this damage is related to mechanical trauma at resonant frequencies. Not many studies have been conducted on humans using allowable levels of vibration. Methodological problems seem to have dissuaded most researchers from making such measures. Most measures of gastro-intestinal activity cannot be made during vibration (Campbell et al. 1989). Kjellberg and Wikstrom (1987) measured electrogastogram (EGG) activity during an experiment designed to assess postural sway. A spectral analysis of the data showed an increase in EGG activity in the 0.05 and 0.13 Hz band when subjects were exposed to vibration at 6 Hz at 1 $m.s^{-2}$.

It is possible that the mechanical effect of vibration on the intestines will increase motility, or movement of ingested material but without appropriate breakdown or absorption taking place. On a long term basis, this may lead to some of the gastrointestinal problems reported in the epidemiological studies. No specific measures of this have been made under vibration, according to the reviewed literature. However, the EGG shows some promise.

Combined effects of noise and vibration

In many environments, especially Armoured Fighting Vehicles (AFV), noise is present as a health and operational hazard with vibration. Cole (1983) presents a review of the combined effects of vibration and noise from a military perspective. A number of the epidemiology papers have also considered both stressors and have suggested that, among other effects, hearing loss due to noise is exacerbated by vibration (Rehm and Wieth, 1984). Chernyuk and Tashker (1989) found that vibration, both whole-body and local, increases sensitivity to noise, and suggested that when these two stressors are present together that the permissible limit for exposure quoted for single stresses should be lowered. Hannunkari, Jarvinen and Partanen (1978) and Heino et al. (1978) reported on the effects of both noise and vibration on locomotive engineers. They found that vibration and noise rated as the highest stresses according to subjective assessments. Guignard (1985) reviewed work undertaken in the U.S.S.R. where they showed that noise and vibration can act additively to cause stress, fatigue and degrade vigilance. Sakakibara et.al. (1989) demonstrated an increase in palmar sweating which appears to be a centralised response rather than locally mediated. The effect was greater with both noise and vibration than with either stressor alone. Manninen (1984) found that systolic blood pressure was higher when exposed to noise and vibration compared to vibration alone. Stressfulness ratings were also increased.

With respect to health implications of an environment where high levels of shock, vibration and noise are present, the effects on hearing show the most potential. Hearing loss can take an acute form which is recorded as a Temporary Threshold Shift (TTS) or a chronic form which becomes a Permanent Threshold Shift (PTS). These changes are usually measured by audiometric methods. It is accepted to date that there is no definitive, consistent relationship between TTS and PTS. However, many researchers still suggest that if a noise is severe enough to cause a TTS, especially one that requires 24 hours or more for recovery, then that noise may well cause PTS if the person is exposed to it on a number of occasions. Allen (1983) provides an insightful review of the effects of vibration and noise on hearing loss and suggests that there is a critical vibration dose above which vibration exacerbates the effects of noise on TTS. The critical dose he suggests is:

$$2g_w^2 t$$

where g_w = g rms weighted by ISO 2631

t = exposure duration in minutes

Seidel et al. (1988) suggest a lower dose between 1 and 2 (100-200 $m^2.s^{-4}$min) than Allen and notes that the minimum dose that may exacerbate TTS will depend on other factors such as

individual sensitivity, temperature, and physical and mental workload.

Many studies where both noise and vibration are present, have not used a control group exposed to only noise at the same intensity, i.e. without vibration. Inclusion of this noise-control group is important to determine the interaction of the two stressors. However, some laboratory studies in this area, especially those by Manninen, have included this control group and found that vibration in combination with noise does increase the TTS produced by noise alone (Manninen, 1986; Manninen and Ekblom, 1984; Manninen, 1984; Manninen, 1985; Manninen, 1982; Manninen, 1984; Manninen, 1983; Manninen, 1983; Manninen, 1988; Seidel et al. 1988; Miyakita, Miura, and Futatsuka, 1987). In the studies undertaken by Manninen, the effects of various forms of both noise and vibration on TTS have been examined. He also included variables such as temperature, physical workload and competition. The combinations of conditions and complex results of these studies are summarised in Appendix B, Table B-13. The difference in TTS_2 between the noise condition and noise plus vibration is shown, along with the different experimental conditions. The experimental design adopted by Manninen resulted in the total exposure time being broken down into 'Sessions'. TTS_2 was measured after each of these Sessions. In most of the experiments, the hearing loss increases over the whole period. Overall, vibration appears to increase the TTS produced by noise. This ranges from a few dB to 13.5 dB depending on the frequency and level of both the noise and vibration, and the other variables used in the studies.

Hughes (1972) conducted the only study reviewed which did not find that noise and vibration together caused TTS even though the noise levels used were 90 and 95 dBA. This conflict with other data may be explained by the short exposure time (25 mins).

Damage to hearing induced by WBV plus noise has been hypothesised as being caused by the same mechanism as Vibration White Finger disease (VWF). Indeed, Pyykkö (1981; 1992) studied hearing loss in lumberjacks and found that those suffering from VWF consistently had a PTS that was 10 dB greater than those individuals who did not have VWF. Vasoconstriction could be produced in the cochlea in the same manner as in the digital blood vessels as a result of sympathetic nervous system activity (1987). Pyykkö, among others, suggests that repetitive sympathetic over-activation can lead to muscular hypertrophy in the walls of peripheral vessels and a decrease in lumen diameter. Noise and vibration presented together as stressors, produce a strong, wideband signal and hence induce a high metabolic demand in the cochlea, which may result in ischemia.

Manninen (1985) provides another explanation of the cause of TTS enhancement. He found that TTS increased along with heart rate and blood pressure, especially in the presence of heat as well as noise and vibration. He suggests that hypertension in the

systemic circulation contributes to changes in the cranial circulation and therefore to the inner ear metabolism. In other studies, he hypothesised that peripheral vasodilation and pooling of blood in the lower extremities may lead to a reduction in blood flow, and therefore oxygen supply to the inner ear, which would result in a disturbance of hair cell function. However, this theory is not supported by experimental data, due to the technical difficulty of determining blood flow to the inner ear. Manńinen (1983) also found that ECG R-wave amplitude decreased with increasing TTS and that a decrease in R-wave amplitude correlates with a decrease in systolic blood pressure. Seidel et al. (1988) suggest another mechanism for TTS caused by the combination of vibration and noise. Vibration and noise stimulate the middle-ear muscles at resonant frequencies, and this stimulation and mechanical stress induce high loads on the inner ear, which exceed those produced during noise exposure alone. This is a more localised mechanism than a global over-activation of the sympathetic nervous system (SNS).

Two animals studies (Hamernik et al. 1980; Hamernik et al. 1981), that combined audiometric and histological techniques, showed not only higher TTS and PTS with a combination of noise and vibration than with either stress separately, but also increased damage to the cochlea. There was a significant loss of outer hair cells with vibration alone without any TTS or PTS seen in the audiometric tests. However, some animals showed no damage to the cochlea although TTS was evident. This further confuses the determination of a mechanism for hearing loss. The authors also draw inferences about the lack of a relationship between TTS and PTS. In this instance, equal TTS at the same frequency did not necessarily lead to the same PTS.

In summary, there is strong evidence to suggest that vibration increases hearing loss in the presence of noise. No work has been found on the effects of repeated shocks, but it is anticipated that the response of the system will be the same, if not worse. Since AFV's produce high levels of vibration, shocks and noise, it is clear that crews are likely to suffer more hearing loss than would be predicted by the noise exposure alone.The measurement of TTS_2 as an index of hearing loss suffers from a number of limitations. Firstly, inter and intra-subject variability is usually high. Variables such as training and adaptation time have to be carefully controlled. TTS_2 also has to be measured within 2 minutes of cessation of the stresses. In this study, there are likely to be a number of demands on the subject at the end of the vibration exposure, given all the other measures that may be taken. This may make the measurement of TTS_2 impractical. The noise levels in AFV's are very high and it is unlikely that for ethical and practical reasons we will not be able to simulate these. This will likely reduce the level of TTS seen post-exposure and it is possible that the magnitude of effect will not be significant. The measurement of TTS should still be considered if the potential problems outlined above can be overcome.

Effect of mental workload

Although this literature review did not set out to cover the effects of vibration and impacts on task performance, as it is outside the original mandate of the current study, some attention has been paid to this area given its potential relevance to the physiological measures as will be discussed below.

Mental workload has been shown to have an effect on physiological response to stress, including that induced by vibration (Harstela and Piirainen, 1985; Chernyuk and Tashker, 1989). Angus et al. (1983; 1987) found that soldiers were more susceptable to the effects of sleep loss if they were working continuously in a command post environment compared with those who only did a few tasks every hour.

Much work has been undertaken on the topic of task performance under vibration with very variable results. The effects of vibration both alone and with noise have been reviewed (Grether et al. 1972; Guignard, 1974). Cole (1983) reviews the effects of noise and vibration on task performance from a military perspective and suggests that the acute effects of vibration on performance will be the limiting factor in the use of AFVs. In general, many of the effects on performance, especially psychomotor tasks, are due to mechanical interference (Gray et al. 1976; Rylands, 1990; Rylands, 1990; Bennett et al. 1978; Bennett et al. 1978). An effect on cognitive performance has not yet been satisfactorily proven. Grether et al. (1971; 1971) found that combined stresses did not produce more effect than single stress alone on performance measures. However, other authors (Champion and Sandover, 1982; Rylands, 1990; Rylands, 1990) have found interactions ranging from no effect, through additive to synergistic.

Ullsperger and Seidel (1980) and Ullsperger, Seidel and Menzel(1986) showed an effect of vibration on auditory evoked brain potentials (AEP) and suggested this as a way of assessing effect on information processing. However, the relationship does not appear to be adequately validated. Chernyuk and Tashker (1989) looked at the effects of both noise and vibration from agricultural machinery on physiology and task performance. The important result from their paper is that the susceptability to the effects of the stressors is related to the mental workload. The authors suggest reductions in noise and vibration permissible limits depending on mental workload.

Landstrom and Lundstrom (1985) used EEG as an indicator of wakefulness and found that vibration increased theta and decreased alpha activity during sinusoidal vibration which supports the theories that monotonous vibration can reduce alertness. However, the effect was not so marked under random vibration

Since we are trying to determine the health effects of operators in an AFV environment, and cannot predict what the effect of undertaking typical crew tasks would be on the physiological response to vibration and repeated impacts, it is important to simulate these. There are a number of psychological test batteries , for example, PETER, and STRES that have been used by military research laboratories to study the effects of stressors on performance (Advisory Group for Aerospace Research and Development, 1989; Guignard, Bittner, and Harbeson, 1983). One of these could be employed to keep the subjects occupied during the experiment's duration.

Conclusions

Vibration and impacts cause a number of physiological changes affecting many of the body systems. Many of the responses to WBV are attributed to stimulation of the sympathetic nervous system. Some of the cardivascular and respiratory responses mimic the effects of moderate exercise. There has been little work on the effects of repeated impacts on physiological parameters. Most of the information reviewed has come from literature on the acute effects of WBV on animals and humans. Throughout this review, a significant problem has been the extrapolation between acute physiological effects and long-term health problems. However, the correlation of biochemical findings and investigations with the physiology shows promise.

Three areas that show the most potential as indices of long term health problems are measures of cardiovascular, gastro-intestinal and hearing performance.

Literature on the cardiovascular response to impacts shows some interesting anomalies. Investigation will include the effect of repeated impacts on the spectral components of the ECG signal and examination of the responsiveness of the cardiac system using phase plots.

Since there is evidence of long term damage to the gastro-intestinal system, the inclusion of an acute measure of the response of this system to vibration and repeated impact is important. The use of EGG to measure gut motility will be investigated along with other transducer systems to assess whole gut movement.

Vibration enhances the effect of noise on hearing loss and since AFVS produce high levels of both stressors, it is important that this health effect is considered. If practical, this study should include noise as a stressor and TTS as a measure.

Biochemical Effects Related to Mechanical Shock and Repeated Impact

The purpose of this review is to investigate disturbances of physiological function, or actual physical damage in humans resulting from exposure to vibration, or repeated impact. Because of the limited experimental data in the whole-body vibration literature, a selection of papers related to localized vibration and exercise were also reviewed. The rationale for this is that localized vibration appears to have overall, systemic effects. Also, the physiological response to vibration is similar to that observed during exercise, and damage reported post-exercise is partly a result of mechanical trauma to tissues due to physical impact.

Many of the studies in the vibration literature involved animal models. In some of the animal studies, the level of vibration was extremely high compared with what a human would be exposed to in a transport vehicle. Physiological and anatomical differences between humans and animals with respect to size, resonant frequency of internal organs, and the response of a quadriped compared with a human in a seated posture also limit direct comparison of data from animal and human experiments.

Biochemical measures in blood and urine are routinely measured in a clinical setting to evaluate stress and strain on the human body. These tests are used to detect physiological and metabolic abnormalities, as well as tissue or organ damage. Careful interpretation of biochemical data can often differentiate between acute and chronic dysfunction. To select appropriate measures to include in the development of a health hazard index in the present study, the biochemical literature is examined in three areas:

- General Stress
- Muscle Fatigue
- Tissue or Organ Damage

General Stress

In response to stress, the homeostatic balance of the human body is disturbed. The response to any general stressor may be assessed by changes in plasma catecholamine concentration, cyclic nucleotides, hormones excreted from the adrenal cortex, brain neurotransmitter alterations, and a general inflammatory response. Several papers which evaluate the effect of vibration in terms of these parameters examine the response to vibration exposure as a form of general physiological stress.

Catecholamines

The activity of the sympathetic nervous system (SNS) is reflected peripherally by plasma catecholamine concentration, and urinary catecholamine excretion. Catecholamine assays are difficult to interpret, in part due to activation of the SNS by many stressors, large inter-individual variations, and low (i.e. nanomolar) physiological concentrations which make the biochemical assay difficult. Catecholamines may be important, however, in the interpretation of cardiovascular, blood flow, and temperature regulation responses to vibration exposure. Catecholamines affect heart rate and the ability of the heart to generate forceful contractions. Catecholamines are also important in regulating blood flow distribution, which in turn can influence temperature regulation.

Elevated sympathetic activation and increased peripheral sensitivity to catecholamines is linked with VWF (Bovenzi, 1986; Bovenzi, 1988; Saito, Inuzuka, and Azuma, 1986; Nakamoto, 1990). As well, changes in catecholamine concentration have been reported in humans exposed to whole-body vibration. Anderson et al. (1977) observed an increased rate of urinary catecholamine excretion following nap-of-the-earth helicopter flying. These data could be confounded by the general stress response to the difficult flying procedures. Kamenskii and Nosova (1989), who selected vibration exposure parameters to simulate modern transport cabs (8-16 Hz, 0.6-1.4 $m.s^{-2}$, 1 hr), observed a 25% decrease in the noradrenaline/adrenaline ratio in the urine, suggesting a relative activation of the adrenal system, a common response to stress. Daleva et al. (1982) also reported an elevated catecholamine excretion rate in tractor-drivers during seasonal field work, although the increase was not significant.

Cyclic nucleotides

The cyclic nucleotides (cyclic AMP, or cAMP, and cyclic GMP, or cGMP) are regarded as markers of autonomic nervous system (ANS) activity, reflecting sympathetic (SNS) and parasympathetic (PNS) receptor functions, respectively. There is not universal agreement in the literature on the effect of vibration on changes in cyclic nucleotides in the blood. Using these markers, Harada et al. (1990) reported that heart rate variations observed in patients with Raynaud's syndrome were related to decreased PNS activity. However, Okada, Ariizumi, and Fujinaga (1991) found no difference in cAMP and cGMP in workers with Raynaud's syndrome. Matoba and Chiba (Matoba and Chiba, 1989), who investigated the effect of whole-body vibration on regional myocardial blood flow (MBF) and cyclic nucleotides in dogs, found an inverse relation between MBF and cyclic nucleotide concentration. It was suggested that increased cyclic nucleotides might be a reaction to decreased MBF due to smooth muscle contraction by vibration. While these data are conflicting and difficult to interpret in the present study, evaluation of cyclic nucleotides might be

useful to compare with alterations in blood flow or other cardiovascular functions.

Glucocorticoids

One of the major systems involved in the maintenance of homeostasis in the body is the hypothalamic-pituitary-adrenocortical axis. The activity of this system can be evaluated by changes in plasma and urinary glucocorticoids. Whole-body vibration exposure in humans at 3 levels of vibration (1-3 Hz, 20 $m.s^{-2}$; 5-7 Hz, 7.5 $m.s^{-2}$; 18-20 Hz, 30 $m.s^{-2}$), for 9 minutes at each frequency range, showed no significant change in 12 hr. urinary excretion of adrenal cortical steroids (Litta-Modignani et al. 1964; Blivaiss, Magid, and Litta-Modignani, 1964). This exposure, although high, was relatively short and may not have provided sufficient stress to elevate adrenocortical activity. In contrast to these data, Dolkas, Leon, and Chackerian (1971) observed a significant elevation of plasma corticosterone in rats immediately following vibration exposure (4.7 Hz, 17 $m.s^{-2}$ for 10 min). In this study, plasma corticosterone remained elevated for 60 min. after vibration exposure. Prolonged elevation of plasma corticosteroids, as observed by Dolkas, Leon, and Cackerrian (1971), is expected as a general stress response. Ariizumi and Okada (1983) also reported a significant increase in plasma corticosterone in rats subjected to high levels of vertical sinusoidal whole-body vibration for 240 min (5 Hz, 20 Hz, or 30 Hz, with 4, 20, or 50 $m.s^{-2}$ acceleration). Plasma corticosterone increased as acceleration increased from 4 to 50 $m.s^{-2}$. It was also significantly elevated, relative to resting concentration, at vibration frequencies from 0.5 Hz to 30 Hz, but there was no dose-response relationship between vibration frequency and plasma corticosterone concentration. The high level of vibration used on the rats in this study is greater than normally experienced in human experimentation or prolonged occupational exposure.

Inflammatory response

A generalized inflammatory response has been linked to the pathogenesis of Raynaud's phenomenon both of occupational and non-occupational origin (Langauer-Lewowicka, 1976). Inflammation is also associated with generalized tissue damage in response to trauma (Kellermann et al. 1989; Koeniger, Hoffman, and Schmid, 1989) and exercise (Smith, 1991). Changes in certain plasma proteins can be measured which indicate an inflammatory response. For example, an increase in beta- and gamma- globulins, and anti-globulin antibodies has been observed in patients with vibration disease, relative to a control population (Langauer-Lewowicka, 1976). In other studies, the presence of an increase in the activity of the enzyme phospholipase A2 has been linked specifically with cardiac and skeletal muscle damage (Jackson, Jones, and Edwards, 1984; Duncan and Jackson, 1987; Duncan,

1988). This enzyme is also thought to play a major role in the etiology of exercise-induced muscle damage, leading to enzyme leakage from muscle tissue (Jackson, Jones, and Edwards, 1984). While the inflammatory response is not specific to vibration, it may be useful to examine some of the typical markers of general/systemic inflammation in the determination of a health hazard index. To date, this has not been measured specifically in relation to whole-body-vibration and repeated impact.

Brain neurotransmitter metabolism

Generalized stress is known to affect central nervous system activity. Ariizumi and Okada (1983) measured changes in certain central neurotransmitters and reported a dose-response increase in whole brain serotonin (5-HT) with higher levels of acceleration. Both brain 5-HT and and its main metabolite, 5 hydroxyindoleacetic acid (5-HIAA) were elevated in rats exposed to whole-body vibration (5 to 30 Hz, and 0.4 to 5.0 $m.s^{-2}$) (Ariizumi and Okada, 1983). The most significant elevation at 20 Hz was thought to reflect a resonant frequency of the rat. In a subsequent study, also in rats, whole brain norepinephrine decreased following 240 min exposure at 20 Hz, 50 $m.s^{-2}$, whereas 5-HT concentration increased, specifically in the hypothalamus (Ariizumi, Yamaguchi, and Okada, 1986). 5-HT has been linked to the regulation of the hypothalamic-pituitary-adrenocortical system. Altered brain neurotransmitter metabolism may be important, therefore, in both neural and endocrine function. However, direct markers of change in central nervous system activity are not attainable in human experimentation.

Muscle Fatigue

Biochemical responses indicating fatigue have been measured in the vibration literature, but more extensively in relation to physical exercise. A biochemical marker of fatigue would be an important factor in a health hazard index, since increased fatigue (monitored as a decrease in metabolic potential, or a decrease in force generated by muscle) will ultimately result in decreased physical and psychological performance, and possibly increased recovery time. Peripheral muscle fatigue has been related to changes in carbohydrate metabolism (lactate, glucose), protein and energy metabolism (ammonia), cortisol, and electrolyte balance (K^+, Mg^{2+}, Ca^{2+}) (Roberts and Smith, 1989).

Lactate

Serum lactate is elevated when aerobic metabolic processes do not produce enough ATP to satisfy energy demand, and anaerobic metabolism is stimulated to contribute to energy production. Generally, increased lactate reflects increased skeletal muscle

activity, and high lactate concentration is associated with peripheral fatigue. Serum lactate was elevated, relative to controls, following nap-of-the-earth helicopter flights, interpreted to reflect increased muscular strain (Anderson et al. 1977). Kamenskii and Nosova (1989) reported an elevated lactate concentration, immediately following whole-body vibration exposure simulated to represent modern transport cabs. Elevation of serum lactate concentration following physical exercise, and presumably following vibration exposure, depends on the intensity and duration of the stressor, individual fitness, fatigue, and muscle fiber composition (Kraemer and Brown, 1986; Long et al. 1990; Farber et al. 1991).

Ammonia

Ammonia is produced during exercise by two predominant pathways: 1. deamination of AMP to IMP, to maintain the energy balance in the cell, and 2. deamination of amino acids which contribute to energy metabolism, particularly branched chain amino acids, during prolonged physical activity. Ammonia has been correlated with fatigue during exercise (Roberts and Smith, 1989), but to date it has not been evaluated in relationship to vibration or impact exposure.

Cortisol

As previously mentioned, the pituitary-adrenocortical system plays a major role in maintaining biological homeostasis in response to various stimuli. Prolonged elevation of cortisol, in the absence of a stressor, is a reflection of chronic adrenal stress, or over activation. Both urinary and plasma adrenal steroid metabolism is altered following whole-body vibration exposure in humans (Litta-Modignani et al. 1964; Blivaiss, Magid, and Litta-Modignani, 1964) and in rats (Dolkas, Leon, and Chackerian, 1971; Ariizumi and Okada, 1983). A model of chronic adrenocortical stress has been developed in relation to overtraining in athletes. Overtraining results in clearly identifiable symptoms of chronic fatigue (Barron et al. 1985; Kuipers and Keizer, 1988). Barron et al. (1985) observed an elevated basal cortisol concentration in overtrained athletes. In addition the normal release of certain hormones in response to insulin-induced hypoglycemia was impaired in the overtrained subject. The impaired response was not directly related to the stress of physical training or racing, since normal athletes (i.e. not overtrained), had a normal endocrine response to an insulin challenge test, even after marathon racing. With respect to the health hazard index, prolonged elevated cortisol would indicate exposure to stress from which an individual was not able to recover on a short term basis.

Glucose

A reduction in blood glucose is frequently used in the exercise literature as a measure of fatigue in prolonged exercise. During short term physical exercise, blood glucose is generally maintained close to its resting concentration. Hypoglycemia often accompanies prolonged moderate exercise (i.e. >1.5 hr), and is related to a reduction in glycogen availability (Kraemer and Brown, 1986; Long et al. 1990). Reduced blood sugar has also been associated with acute exercise-induced fatigue (Roberts and Smith, 1989) and overtraining (Barron et al. 1985).

While prolonged exercise provides a useful model of hypoglycemia, the effect of vibration exposure on blood glucose concentration is of greater interest in the present study. Maintenance of blood glucose is particularly important in an occupational setting, to prevent sympoms of hypoglycemia which can interfere with task performance. A single four hour exposure to low amplitude, high frequency whole-body vibration in rabbits, and in dogs, results in a decrease in blood glucose and glycogen (Sinitsyn, Rumyantsev, and Voronova, 1964). The observed decrease was more pronounced following repeated exposure to vibration. In contrast to this study, Dolkas et al. (1971) reported an increase in plasma glucose in rats following shore term exposure to whole-body vibration (4.7 Hz, 17 $m.s^{-2}$ for 10 min.). Because of the great difference in vibration exposure, it is difficult to compare these results. No papers were reviewed which measured the effect of vibration and impact exposure on blood glucose in human subjects.

Electrolytes

Electrolyte balance is important for maintenance of many physiological and biochemical responses. A small change in serum potassium (K^+), or serum sodium (Na^+) may alter resting membrane potential. Recently, elevated serum magnesium (Mg^{2+}) concentration has been related to the development of muscular fatigue. A change in calcium ion (Ca^{2+}) concentration may also be related to physiological changes, and contribute to the initiation of tissue damage, or fatigue. Because of their diverse physiological functions, a change in the concentration of serum electrolytes may be important in the development of a health hazard index.

Potassium

Potassium is released from skeletal muscle during muscular contraction, and is returned to the cell by the action of the sodium-potassium (Na+-K+) pump. The activity of the Na+-K+ pump is not sufficient to maintain low extracellular K+ during physical activity (Clausen, Everts, and Kjeldsen, 1987). Consequently K+ accumulates in the extracellular space, and

spills into the blood. Potassium has been investigated relative to its affect on membrane potential, ventilation, and on electromyograph (EMG), and electrocardiograph (ECG) signals. An increase in extracellular K+ concentration from 4.0 mM to 5.5 mM would decrease the transmembrane potential of a cell from -95 mV to -85 mV (based solely on the Nernst equation), which would significantly increase the sensitivity of all electrically active tissue (nerves, cardiac and skeletal muscle) (Linton et al. 1984; Sjogaard, 1986; Sjogaard, 1990; MacLaren et al. 1989). Increased serum potassium has also been correlated with excessive ventilation during exercise, presumably by increasing the discharge of peripheral chemoreceptors in the carotid body (Paterson, 1992). A potassium-induced depolarization of membrane potential may also affect EMG signals (Sjogaard, 1986; Sjogaard, 1990). Activity of cardiac muscle is also affected by elevated serum potassium, and this can be detected on a standard ECG. Increased serum potassium results in a characteristic "P wave" elevation in the ECG (Kjeldsen, 1991), although a dose-response relationship between serum potassium and either "P wave" height or area has not been established.

The effect of increased serum potassium may not be completely negative. Localized potassium release may also increase local vasodilation within the contracting muscle (Lindinger and Sjogaard, 1991), which could increase delivery of substrates and enhance removal of waste products from working muscle. However, while this increase in local blood flow would benefit working muscle, blood would have to be diverted from other areas of the body, possibly through sympathetic vasoconstriction.

There are few reports in the literature on the effect of vibration exposure on serum potassium. Prolonged repeated exposure to whole-body vibration (12 Hz, 15 $m.s^{-2}$, 5 $hr.day^{-1}$, 130 hr) has been reported to result in increased serum potassium in monkeys (Badger et al. 1974). This study did not relate the change in serum potassium to any physiological parameters.

Magnesium

One recent paper suggested that elevated magnesium ion (Mg^{2+}) concentration in skeletal muscle may contribute to decreased muscular endurance, and the onset of fatigue (Madsen et al. 1992). By increasing Mg^{2+} concentration, intracellular Ca^{2+} release is reduced from the sarcoplasmic reticulum (SR). It has been hypothesized that since Ca^{2+} is obligatory for both contraction and relaxation in muscle, Mg^{2+} inhibition of Ca^{2+} release could contribute to fatigue in prolonged exercise. Madsen et al. (1992) reported a significant elevation of muscular Mg^{2+} concentration at exhaustion in both trained and detrained individuals. Since whole-body magnesium should not change during activity, it is possible that an increase in intramuscular Mg^{2+} concentration may be reflected by a decrease in serum Mg^{2+}

concentration, although this hypothesis would need to be verified.

In the vibration literature, only one study was reviewed which measured serum magnesium concentration. Kosmakos, Keller, and Collins (1975) found that serum magnesium concentration in rats was unchanged immediately following 4 minutes vibration at 3 frequency ranges from 12 to 211 Hz, although Mg^{2+} was consistently lower 1 day following vibration treatment. While it is difficult to extrapolate these data to prolonged whole-body vibration, it will be useful to measure serum magnesium concentration as another indication of muscular fatigue, in conjunction with EMG data.

Calcium

Calcium ion (Ca^{2+}) concentration is highly regulated in the body under normal situations. It is involved with several metabolic and physiological processes, and is essential for normal muscle and nerve activity. During physical exercise, there is normally little change in serum Ca^{2+} concentration (Long et al. 1990). A change in Ca^{2+} concentration has been reported, however, in response to vibration exposure. Badger (1974) observed reduced serum Ca^{2+} concentration in monkeys following prolonged vibration exposure (12 Hz, 15 $m.s^{-2}$, 5 $hr.day^{-1}$, 130 hr). No explanation was suggested for this, but it could potentially be related to a mechanism of muscle fatigue. Elevated intracellular calcium may be important in the etiology of local skeletal muscle damage (Jackson, Jones, and Edwards, 1984; Duncan and Jackson, 1987; Duncan, 1987; Duncan, 1988), and subsequent release of skeletal enzymes (LDH, CK) to the circulation (Jackson, Jones, and Edwards, 1984).

Tissue or organ damage

Exposure to vibration and mechanical shock has been associated with an increased risk of internal organ or tissue damage. Damage to internal organs is thought to be most severe at the resonant frequency of individual organs, due to alternate compression and stretching of internal organs in the abdominal and thoracic cavities. Animal studies have shown evidence of altered physiological and structural damage to heart (Megel et al. 1962; Megel et al. 1963; Aria, Onozawa, and Iwata, 1990), lung (Megel et al. 1963; Inaba and Okada, 1988; Aria, Onozawa, and Iwata, 1990), brain (Megel et al. 1963), kidney (Megel et al. 1962; Megel et al. 1963; Boorstin, Hayes, and Goldman, 1966; Sackler and Weltman, 1966), gastro-intestinal (GI) tract (Megel et al. 1963; Badger et al. 1974), liver (Cope and Polis, 1959; Mandel, Robinson, and Luce, 1962; Sackler and Weltman, 1966; Ivanovich, Antov, and Kazakova, 1981; Ivanovich, Antov, and Kazakova, 1977; Aria, Onozawa, and Iwata, 1990), skeletal muscle

(Aria, Onozawa, and Iwata, 1990), adrenal glands (Megel et al. 1962), and reproductive organs (Sackler and Weltman, 1966).

Studies involving humans cannot directly assess internal organ damage, although evidence of internal damage is inferred by data reported in the epidemiological literature. In some instances, biochemical markers measured in blood, urine, and feces, have been used as evidence of altered internal organ function or actúal physical damage.

Strenuous physical exertion also results in tissue or organ damage, which is often related to mechanical impact. As a result, data from the exercise literature may provide guidelines to use in the assessment of the effect of vibration and mechanical shock on the human body.

Organ and cell membrane structure

Damage to organs and cell membranes may result from physical tearing of tissue, or from more subtle changes to the composition of the membrane structure. After vibration exposure at 50 and 150 Hz, Tzvetkow and Tzanev (1991) found significant changes in the fatty acid composition of lipids in the membranes of organs and tissues, and in the phospholipid structure of the erythrocyte membranes. Changes in the fatty acid moiety of the lipid were apparent in the length of the carbon chain, and number of double bonds. Changes in the fatty acid composition of the membrane lipids in liver were more evident, followed by those of the kidney. A change in the structural composition of tissue membranes may explain some of the results of tissue or organ damage following exposure to vibration.

Endothelial damage in blood vessels

Many studies have linked the etiology of vibration white finger to structural damage of the endothelial layer of the wall of arteries and arterioles in peripheral vessels repeatedly exposed to vibration (Okada, 1969; Okada et al. 1987; Nerem, 1973; Blunt et al. 1980; Cimminiello et al. 1991). Nerem (1973; 1977) concluded that vibrating tools produce sufficient shear stress to damage the endothelial cells in the arterial wall. A model proposed by Okada et al. (1987) suggests that damage to the endothelial layer of the arterial wall induced by local vibration causes a series of changes in the blood vessel wall (such as endothelial desquamation, platelet aggregation, microthrombi formation), including the induction of smooth muscle cells, which migrate from the media to the intima of the arterial wall. Proliferation of smooth muscle in the intimal layer results in intimal thickening, and a reduction in the interior diameter of the blood vessel, thereby affecting blood flow.

Recently, Jayson et al. (1991) proposed that low back pain associated with exposure to whole-body vibration may be due to vascular endothelial damage. They found that the concentration of von Willebrand's Factor (vWF) antigen in serum was significantly higher in humans after 25 minutes of exposure to vibration at 5 Hz (ISO 2631 Fatigue Decreased Proficiency limit). Increased serum vWF has also been reported in patients with Raynaud's syndrome (Ikehata et al. 1980; Cimminiello et al. 1991). Von Willebrand's Factor is frequently used to assess vascular damage in clinical settings, ranging from systemic vasculitis to diabetic microangiopathy (Pasi et al. 1990; Bleil et al. 1991; Castillo et al. 1991; Porta, La Selva, and Molinatti, 1991). If vascular damage is occurring in response to whole-body vibration, measurement of von Willebrand's Factor will be a useful measure to include in the health hazard assessment.

Blood viscosity

Increased whole blood viscosity has been related to the etiology of vascular disease in primary, as well as vibration-induced, Raynaud's syndrome. (Pringle, Walder, and Weaver, 1965; Blunt et al. 1980; Ikehata et al. 1980; Okada et al. 1987; Okada, Ariizumi, and Fujinaga, 1991). Increased blood viscosity may impede microcirculation in the capillary beds, and increase the risk of damage to the endothelial layer of the blood vessel wall (Wood, Doyle, and Appenzeller, 1991). It has been suggested that changes in the viscosity of blood are related to the degree, rather than the cause of vascular endothelial damage (Blunt et al. 1980). In all of the papers on this topic, an association has been made between patients with Raynaud's syndrome and increased blood viscosity. However, the data are cross-sectional, and do not exclude the possibility that individuals with higher initial blood viscosity would be predisposed to vascular damage leading to clinical symptoms of Raynaud's syndrome (Pringle, Walder, and Weaver, 1965; Blunt et al. 1980; Okada, Ariizumi, and Fujinaga, 1991). In light of this suggestion, it may be prudent to: 1. screen individuals who will be exposed to repeated, prolonged vibration for high blood viscosity (Pringle, Walder, and Weaver, 1965); and 2. ensure adequate hydration of individuals to eliminate an acute increase in blood viscosity resulting from dehydration (Long et al. 1990; van Beaumont, Underkofler, and van Beaumont, 1981).

Erythrocyte hemolysis

Impact associated with running has been related to erythrocyte damage in a condition termed "foot strike hemolysis" (Miller, Pate, and Burgess, 1988). It is detected by a fall in hemoglobin concentration, and a concomitant decrease in free haptoglobin (which binds free hemoglobin to carry iron to the liver for recycling) (Eichner, 1985; Eichner, 1986). Research has also shown that some intravascular hemolysis may occur in distance

swimmers, with no impact (Eichner, 1986). It was suggested that turbulance generated in small blood vessels in vigorously exercising muscles is sufficient to cause hemolysis. Erythrocyte damage may also be detected by the presence of hemoglobin in the urine, or hemoglobinuria (Stipe et al. 1984; Eichner, 1986). Measurement of hematocrit, hemoglobin, haptoglobin, and the presence of hemoglobin in urine would confirm hemolysis caused by whole-body vibration.

Bone and joint damage

In humans, occupational exposure to vibration, mechanical shock and impact has often been linked with bone and joint dysfunction, particularly in the spine (Rosegger and Rosegger, 1960; Guignard, 1972; Radin et al. 1973; Westgaard et al. 1986; Froom et al. 1986; Guidotti and Cottle, 1987; Sandover, 1988). Whole-body vibration has been identified by several investigators as an etiological factor for low back pain (Bowden, 1986; Pope et al. 1984; Pope, Wilder, and Frymoyer, 1980; Wilder et al. 1982; Troup, 1978; Troup, 1985). Biochemical and morphological changes in the dorsal root ganglion of rabbits after low frequency whole-body vibration provides objective evidence of a relationship between back pain and vibration, since there is an increase in the production of neuropeptides (substance P, VIP) associated with pain (McLain and Weinstein, 1991; Weinstein et al. 1987). Holm and Nachemson (1985) used an animal model (anaesthetized pigs, vibrated in an upright position at 4-9 Hz for 1-6 hr.), to determine the effect of vibration on nutrient availability to the spine. Post-vibration exposure, a decreased uptake of radioactive tracer and water into the discs supplying nutrients to the vertebral column was observed. Reduced nutrient availability could potentially lead to degeneration of the intervertebral discs.

Damage to connective tissue in joints can be detected by an increase in hydroxyproline (a chemical found almost exclusively in collagen of connective tissue) excretion in urine. Kasamatsu et al. (1982) observed a significantly greater urinary excretion of hydroxyproline in workers exposed to occupational hand-arm vibration. If joint injury is suspected as a result of whole-body exposure to vibration, urinary excretion of hydroxyproline may be a useful indicator.

Gastro-intestinal tract

Evidence of gastro-intestinal (GI) tract lesions may be detected by the presence of fecal hemoglobin. Presence of blood in feces has been observed in monkeys exposed to high levels of vibration (12 Hz, 15 $m.s^{-2}$, 5 hr. daily, for a total of 130 hr.). Within 2 days after vibration began, tests were positive for blood in stools (Badger et al. 1974; Sturges et al. 1974). Elevated fecal hemoglobin has also been reported following

strenuous physical activity in humans (Potera, 1984; Stewart et al. 1984; Fisher et al. 1986; Kehl et al. 1986). It is frequently assumed that the impact associated with physical activity such as running is responsible for gastro-intestinal lesions. However, GI lesions have been reported following strenuous physical activity without impact, such as cross-country ski racing (Kehl et al. 1986). It has been suggested that a reduction in blood flow to the GI tract, as a result of shunting blood flow to the active peripheral muscles, contributes to GI tract lesions during strenuous physical activity. This has been confirmed by a reduction in blood flow to internal organs measured immediately following exercise in athletes experiencing GI tract lesions (Kehl et al. 1986).

As mentioned in the Physiology and General Stress section, an increased sympathetic nervous system response to vibration and shock could alter blood flow distribution and reduce blood flow to internal organs, in a similar manner that occurs during strenuous exercise (Stewart et al. 1984). This could then potentially cause local hypoxia, leading to damage of the GI tract (or other internal organs). Megel et al. (1963) also suggested that local hypoxia was a major contributor to internal organ damage during vibration stress in rats. An increased frequency of injury to internal organs (including GI tract and stomach) was observed at altitudes greater than 10,000 feet as a result of high frequency, low amplitude vibration exposure. The greater incidence of injury at altitude was attributed to a reduced partial pressure of oxygen, increasing the local hypoxia created by a reduction in blood flow to internal organs (Megel et al. 1963). It is possible that vibration and impact may increase the incidence of GI bleeding when blood flow to the internal organs is already reduced.

Liver

Damage to liver by vibration exposure has been implicated in studies reporting increased serum enzyme activity (Cope and Polis, 1959; Mandel, Robinson, and Luce, 1962). Tambovtseva (1968) reported chronic changes in serum protein content, protein fractions, and albumin/globulin ratios (reflecting liver damage) in excavator operators. In this study, the workers were exposed to occupational vibration for a minimum of 5 years. In addition, Ivanovich, Antov, and Kazakova (1977; 1981) studied the biochemical and histological changes to liver excised from animals after exposure to high frequency vibration, 2 hr./day for 10 days. They found that markers of aerobic metabolism (succinate dehydrogenase (SDH) and ATPase) were significantly decreased, whereas markers of anaerobic metabolism (glucose 1,6 phosphate dehydrogenase (G1-6-PDH) and lactate dehydrogenase (LDH)) increased in liver homogenates. Damage to cellular organelles were observed by electron micrographs (including enlarged, elongated mitochondria with reduced inner membranes (cristae), sarcoplasmic reticulum with enlarged cisternae, and

large areas of glycogen within cells) (1977; 1981). Biochemical and histological changes to cells were more pronounced following combined exposure of the animals to noise, heat, and lead, in addition to vibration, than exposure to any single factor (Ivanovich, Antov, and Kazakova, 1981).

Skeletal and cardiac muscle

Strenuous physical activity is often associated with the leakage of small molecular weight proteins from striated muscle into the circulation. This is usually detected by the presence of the enzymes lactate dehydrogenase (LDH) and creatine kinase (CK), or the presence of myoglobin in the circulation (Kraemer and Brown, 1986; Norregaard Hansen et al. 1982; Apple and Rhodes, 1988; Long et al. 1990; Dressendorfer et al. 1991). One study has reported elevated serum LDH and CK in humans following exposure to vibration (Anderson et al. 1977). Immediately following nap-of-the-earth helicopter flights in this study, both LDH and CK were elevated, but the elevation was within normal values.

In some studies of elevated serum enzymes, histological evidence of mechanical trauma to tissue is present, and physical disruption of the muscle cell membrane has been observed. However, other studies have shown that mechanical trauma is not required to induce the release of muscle enzymes to the circulation. Enzymes are not released to the circulation immediately. Release tends to reach a peak at 24-36 hours post-exercise (Apple and Rhodes, 1988). A similar observation of a delayed release of muscle enzymes has been reported post-myocardial infarction (Roberts, Henry, and Sobel, 1975; Willems et al. 1985; Shibata et al. 1985). An increased concentration of circulating muscle enzymes have also been reported following strenuous physical activity in rowers (Norregaard Hansen et al. 1982), and cyclists (Hortobagyi and Denahan, 1989), both non-impact sports. Thus the temporary muscle damage may occur following strenuous physical activity without physical impact. This implies that other models of membrane damage may be important in the etiology of enzyme leakage, such as Ca^{2+}-induced damage to the muscle membrane (Jackson, Jones, and Edwards, 1984; Furuno and Goldberg, 1986; Duncan, 1987; Duncan and Jackson, 1987; Duncan, 1988). In addition to skeletal muscle damage, there is evidence that muscle-specific enzymes may also be released from cardiac tissue to the circulation during strenuous activity (Norregaard Hansen et al. 1982). At this time, the pathological implication of this is unknown.

Elevated serum enzyme activity has been used to estimate the area of tissue damage resulting from myocardial infarction (Norris et al. 1975; Roberts, Henry, and Sobel, 1975; Shibata et al. 1985; Willems et al. 1985). As an extension of these methods, Apple and Rhodes (1988) assessed the area of skeletal muscle damage size (SMDS) following exercise. Their equation is:

$$SMDS = (BW)(K)(CKr)$$

where BW is body weight, K is a constant. CKr is the relative amount of creatine kinase (CK) released to the circulation, and includes assumptions about CK distribution, uptake, and degradation. In spite of the assumptions, however, the results provide a reasonable estimation of muscle damage, and a similar principle may be useful to assess tissue damage in response to whole-body vibration and repeated impact.

Conclusions

The inclusion of a biochemical marker in the development of a health hazard index for exposure to whole-body vibration and repeated impact would provide an objective index for health assessment. The goal of the health hazard index is to reduce vibration and impact exposure below a level which may cause physical damage or disability. Thus, biochemical evidence of severe physiological disturbance, and tissue or organ damage, would confirm that a "safe" exposure limit has been exceeded.

There are obvious limitations in many of the studies cited in the review of biochemical markers related to vibration, shock, and impact. The first is the breadth of the data base. In many, only cross-sectional data were reported, with no appropriate control group, which limits any interpretation of a cause-and-effect relationship. Other studies report clinical or field trial data in workers exposed to vibration in an occupational environment, without a controlled experimental intervention. With respect to organ and tissue damage, studies on animals cannot be directly extrapolated to human response. Animals will have a different response to vibration as a result of a difference in body size, resonant frequency of internal organs, and their quadripedal posture. In addition, some of the health hazards of vibration and impact exposure may develop only after prolonged or repeated exposure (in some instances, after years of occupational vibration exposure). However, in spite of these limitations, there seems to be adequate evidence in the literature to justify pursuing a biochemical marker indicative of vibration-induced damage in humans. The data may also provide a useful compliment for other physiological parameters monitored (i.e. ECG, EMG).

The selection of appropriate tests for this study will be limited, in part, by expense and technical difficulty. Care must also be taken to standardize timing and method of collection, preparation, and storage of biological fluids, since many are extremely labile, and may have to be processed or frozen rapidly for future analysis. As well, it is strongly recommended that any single analysis is carried out in the same laboratory throughout the study, to reduce problems with inter-assay variation.

One factor which must be considered is correction of all biochemical data for hydration state and fluid shifts which may be a consequence of vibration exposure. For example, without appropriate correction, an apparent increase in concentration of a serum metabolite might merely reflect hemoconcentration resulting from fluid shifts, dehydration, or postural changes.

In conclusion, the most useful indicators of physiological and biochemical disturbances induced by whole-body vibration and impact will be the measurement of serum electrolytes, lactate, ammonia, glucose, cortisol and catecholamines. Indicators of tissue and organ damage, which are feasible in this study could include serum LDH and CPK activity, von Willebrand's factor, free hemoglobin, haptoglobin, myoglobin, viscosity, and hematocrit. Urinary and fecal measurements which would be useful would include urinary corticosteroids, hemoglobin and myoglobin, and fecal hemoglobin. Collection of urine and feces may be more difficult, and could potentially affect subject compliance to the experimental protocols. This list is not final, and data from the pre-pilot and pilot studies will contribute to the final selection of analyses to be completed.

With careful planning, the data collected as biochemical markers should provide a useful addition to the development of a health hazard index for human exposure to vibration, mechanical shock, and repeated impact.

Muscle Response to Vibration

Introduction

Electromyography (EMG) is a method of measuring depolarization of muscle cells, which precedes the generation of tension by those cells. EMG can therefore be used to monitor various aspects of muscle function. The response of paraspinal muscles to whole-body vibration and impact has been studied using EMG to assess localized muscle fatigue (Hansson, Magnusson, and Broman, 1991; Hosea et al. 1986; Magnusson, Hansson, and Broman, 1988; Robertson and Griffin, 1989; De Luca, 1985; Seidel, Bluethner, and Hinz, 1986; Wilder, Frymoyer, and Pope, 1983), phase and timing relationships between muscle response and acceleration (Hagena et al. 1986; Robertson, 1987; Robertson and Griffin, 1989; Seidel, 1988; Seidel, Bluethner, and Hinz, 1986; De Luca, 1985), and to estimate compressive loading and torque about the spine (Marras and Mirka, 1991; Ortengren, Andersson, and Nachemson, 1981; Seidel, Bluethner, and Hinz, 1986; Seroussi et al. 1987; Seroussi, Wilder, and Pope, 1989; Wilder et al. 1990). In general, these parameters are of interest because of their association with stabilization of the spine, and their subsequent association with back pain and injury to spinal tissues.

Muscle fatigue may diminish the ability of muscle to adequately compensate for perturbing forces, while out-of-phase or untimely muscle response can contribute to postural destabilization and increase both torque and compressive loading of the spine (Seroussi, Wilder, and Pope, 1989; Seroussi et al. 1987). However, no direct cause-effect or dose-response associations have been made between muscle fatigue or timing of response and either postural destabilization or enhancement of forces at the spine. Considerable effort has been directed at characterizing the association between fatigue and vibration characteristics, timing and vibration frequency, as well as the muscular contribution to torque or load at the spine.

EMG Methods

The use of needle, wire, or surface electrodes each has inherent advantages and disadvantages. Needle electrodes and wire electrodes have higher impedance than surface electrodes, leading to a greater voltage drop at the electrode-tissue interface, and a consequential loss in signal power. In addition, the insertion of needle or wire electrodes can be uncomfortable for the subject. However, they provide an EMG signal that represents a more focussed region of muscle and is thus less likely to be contaminated by neighbouring muscles compared with surface EMG. This makes needle or wire electrodes

the preferred choice for analyzing single motor units or when a specific depth and region of a muscle are to be studied. Wire electrodes are subject to migration and therefore make reproducibility a potential problem; however, they are less painful than needle electrodes. Surface electrodes are easy to apply, have low impedance, and provide a more general EMG signal representative of the average response of a large area of muscle (Basmajian and De Luca, 1985). For these reasons, surface electrodes have been almost exclusively used in studies of muscle response to whole-body vibration and impacts. The invasiveness and potential for injuring a subject with needle or wire electrodes during vibration and impacts are additional reasons for the selection of surface EMG techniques to study the muscle response to whole-body vibration and impacts.

Conditioning of EMG signals is dependent upon the type of analysis required and the myoelectric frequency range of interest. Myoelectrical activity predominantly spans 20 to 250 Hz when surface electrodes are used, but has motor unit action potential (MUAP) components as high as 10 kHz (Basmajian and De Luca, 1985). Typical sampling rates for EMG range from 500 Hz, for analysis of surface EMG, to as high as 50 kHz, for MUAP decomposition using needle electrodes. The sampling rate must be at least twice the highest frequency in the signal analysed; however, sampling at several times the highest frequency is preferred (Basmajian and De Luca, 1985). Low pass filtering at half the sampling rate is used to prevent aliasing and to remove high frequency noise. High pass filtering at approximately 20 Hz prevents motion artifact and eliminates unstable low frequency EMG components (Basmajian and De Luca, 1985). Most methods of EMG time-domain analysis require prior removal of direct current bias and full wave rectification of the signal.

Motion artifact is a problem of particular concern in studies of the muscle response to vibration or impacts. The rectified average EMG has been used to identify signals with an amount of motion artifact considered to unacceptably contaminate the EMG signal (Robertson, 1987; Robertson and Griffin, 1989). These signals can then be excluded from further analysis. The rationale for using the rectified average EMG to identify motion artifact is as follows: since EMG signal amplitude is evenly distributed about an isoelectric potential, any deviation from baseline in the rectified average is deemed motion artifact. Careful instrumentation of the subject is also important to minimize motion artifact at source.

Noise from 60 Hz electrical interference can be minimized through isolation and shielding of system components, adaptive filtering (Iyer, Ploysongsang, and Ramamoorthy, 1990), differential amplification, the application of a miniature preamplifier in close proximity to the recording electrodes, and notch filtering at 60 Hz The use of a notch filter, however, also eliminates a large component of the EMG signal at 60 Hz, and is therefore the least preferable of the above methods. Fiber

optics and telemetry have been recently employed to minimize induction of electrical interference by recording cables, thereby allowing the data acquisition system to be located some distance from the subject.

Elimination of ECG components from EMG signals has proven to be a non-trivial matter when EMG electrodes are placed on the trunk or neck. Seidel and Pietschmann (1979) used an acquisition-off switch triggered by the onset of the R-peak and with a reset delay equal to the Q-T interval to automatically eliminate portions of the EMG signal corresponding to the Q-T interval. Other automated systems of identifying unacceptable parameters have been developed for specific applications where certain restrictions can be placed on the expected EMG signal (Arvidsson, Grassino, and Lindstrom, 1984). An alternative approach has been to manually process small segments of EMG deemed to be free of QRS complex, and to eliminate P and T waves by setting the high-pass filter cutoff at 20 Hz (Javaheri et al. 1987). Robertson and Griffin (1989) present extensive discussion describing development of methods and artifact elimination during their studies on whole-body vibration and impacts. ECG was not deemed by them to be a significant artifact, since the average rectified ECG was insignificant in magnitude relative to the corresponding average rectified EMG in the same time interval.

Muscle Fatigue

Muscle fatigue has classically been defined in terms of a reduced ability to produce contractile force (De Luca, 1985). Other definitions of fatigue are based on the development of muscle pain, muscle tremor, alterations in EMG parameters, and subjective response (De Luca, 1985; Bigland-Ritchie, 1984; Bigland-Ritchie and Woods, 1984; Magnusson, Hansson, and Broman, 1988; Moussavi et al. 1989; Walsh, Ito, and Grassino, 1991). In essence, muscle fatigue is a time-dependent process that leads to failure of the system (De Luca, 1985). From this perspective, pain, alterations in EMG parameters, and subjective evaluation provide information regarding the time-dependent fatigue process, while a reduction in contractile force generation defines failure of the system. De Luca (1985) demonstrated that EMG parameters indicate fatigue long before generated force declines in a sustained isometric contraction. This is interpreted to mean that metabolic fatigue precedes contractile fatigue. EMG measures can therefore provide objective evidence of muscle fatigue prior to contractile failure.

The mechanism of local muscle fatigue is still a matter of great debate and most likely involves a complex interplay between metabolic, circulatory, and contractile factors, whose relative contributions depend upon the circumstance leading to fatigue (De Luca, 1985).

EMG Markers of Localized Muscle Fatigue

Several methods of processing EMG data have been developed in an attempt to quantify localized muscle fatigue. These include time domain analyses to characterize integrated EMG (IEMG) (Berthoz, 1972; Moritani, Nagata, and Masuo, 1982), rms amplitude (Arendt-Nielsen and Sinkjaer, 1991; Seidel, Beyer, and Brauer, 1987), and amplitude probability distribution (Jonsson, 1988); as well as frequency domain analyses to characterize median frequency (Arendt-Nielsen and Sinkjaer, 1991; Walsh, Ito, and Grassino, 1991), mean frequency (Hagg, 1991), centre frequency (Luciani et al. 1983; Hagg, 1991; Pope et al. 1985; Pope et al. 1984), and high/low frequency band ratio. More complex analyses has been used to determine decline in muscle conduction velocity through the normalized ratio of the first and zero order spectral moments (Lindstrom, Magnusson, and Petersen, 1970). Time domain analyses have also been utilized to quantify frequency-related parameters, such as zero-crossing rate (Hagg and Suurkula, 1991) and autoregression approximations of modal frequency (Bower et al. 1984; Kihwan and Minamitani, 1991).

Metabolic muscle fatigue is indicated in the above methods of analysis by an increased EMG amplitude, spectral compression, a decrease in the characteristic frequencies, and a decrease in conduction velocity. The change in these parameters can largely be explained by the incorporation of low frequency components which add to the total power of the EMG signal and place greater weight on the low frequency spectrum. Motor unit recruitment, motor unit synchronization, changes in muscle fibre conduction velocity, and rate of motor unit discharge have been proposed as physiological mechanisms that could account for the increased EMG amplitude and spectral changes (De Luca, 1985).

De Luca (1985), in a critical review of the literature, stated that the preferred method for frequency shift was to characterize mean and median frequencies, whose decline is linearly related to the average conduction velocity of the muscle fibres. Merletti, Knaflitz, and De Luca (1990) found the mean and median frequency parameters to be more sensitive than amplitude measures for monitoring fatigue. Andersson, Ortengren, and Herberts (1977) found the variance in power spectral density to be greater than that for full wave rectified amplitude in assessing back muscle fatigue; however, changes in these two parameters correlated well with each other. Seidel et al. (1985) found that EMG amplitude changes were inferior to autoregressive determination of modal frequency for quantification of low back muscle fatigue at low force levels. Gogia and Sabbahi (1990) report the median frequency to be a reliable objective measure of cervical paraspinal muscle fatigue.

The same EMG data can be analysed using various methods, therefore a decision to monitor a single marker of muscle fatigue

is not necessary. Both time and frequency domain analyses can be individually applied to the same EMG data.

Paraspinal EMG Fatigue Parameters and Back Pain

The use of paraspinal EMG as a tool to identify and assess back pain has been explored largely because of the difficulty in diagnosing back pain through subjective methods. There are two primary theoretical models linking paraspinal EMG and chronic low back pain: the stress-causality model and the reflex spasm model (Nouwen and Bush, 1984). The stress-causality model relates increased muscle activity to ineffective stress management. An increased reactivity of paraspinal muscles to psychological stress is predicted in patients already suffering from low back pain, since pain itself is a contributing stressor. The reflex spasm model suggests the existence of a muscle spasm reflex which serves to immobilize an area with organic pathology. Thus there is a coincident relationship between pain and increased muscle activity, but no causal relationship. The reflex spasm model also suggests that increased paraspinal EMG may reflect local tissue damage. While the basic premise differs in the two models, the outcome is the same. Paraspinal EMG should be greater in low back pain patients, both at rest and during movement. Nouwen and Bush (1984) reviewed the literature and found no consistent trend to support the assumption that low back pain patients have greater EMG activity. Beidermann (1991), however, suggests that previous research attempting to link paraspinal EMG parameters and back pain have used correlational procedures which yield inappropriately low reliability coefficients, and therefore lead the authors to incorrectly reject the method.

Roy et al. (1989) have since developed a Back Analysis System which discriminates reliably between chronic low back pain sufferers and controls by monitoring paraspinal EMG at L5 during isometric contraction. During fatiguing test contractions, the initial median frequency was found to be lower and the fatigue-induced decline in median frequency was steeper in patients than in controls. Both low back pain and control subject groups demonstrated bilateral asymmetry, indicating that asymmetrical muscle activity was not contributing significantly to the incidence of low back pain. In subsequent papers (Klein et al. 1991; Roy et al. 1990), these authors were able to accurately classify university rowers as suffering from low back pain or being pain free, and demonstrated that their Back Analysis System was more sensitive and specific than traditional range-of-motion methods of diagnosis. While the technique is relatively novel, it suggests that spectral characteristics of paraspinal EMG, which are related to fatigue and fatigability, may be of use in objectively identifying chronic low back pain.

Hagg and Suurkula (1991) used zero-crossing rate during short test contractions and during an endurance test in a longitudinal study of assembly workers to predict work-related myalgia in the neck and shoulders. They found that the absolute crossing rate and the time constant for decline in zero crossing during the endurance test were significantly related to myalgia. The authors concluded that their method held no predictive value but that it could be useful as a diagnostic tool.

WBV and Paraspinal Muscle Fatigue

Fatigue of paraspinal muscles under conditions of vibration is somewhat different from the classical muscle fatigue described during sustained contractions. During whole-body-vibration, induced muscle forces are usually very small, evidence of muscle fatigue is pain rather than diminution of force output, and fatigue takes longer to be evidenced than in sustained contractions (Bowden, 1986).

There is some controversy as to whether constrained sitting postures or vibration are responsible for the postural muscle fatigue and back pain experienced by helicopter pilots and other heavy equipment operators. Both vibration and static seated postures have been demonstrated to increase paraspinal muscle activity (Robertson, 1986; Robertson and Griffin, 1989; Seroussi, Wilder, and Pope, 1989; Pope, Wilder, and Donnermeyer, 1986).

Research on the effects of helicopter vibration have shown that the level of postural muscle fatigue, as evidenced by EMG parameters, and subjective fatigue response using visual analog scales, increases with continued exposure to whole-body vibration (Bowden, 1986; Pope et al. 1985). Bowden (1986) notes that there is a strong association between helicopter pilots' back pain and flight duty; however, both postural and vibrational constraints of the cockpit may contribute to increased muscle activity and consequential fatigue. Wilder et al. (1982; 1983) determined that a 30 minute seated exposure to sinusoidal whole-body vibration at 1 to 20 Hz produced muscle fatigue (EMG spectral shift) while no muscle fatigue was seen in a similarly seated, non-vibrated condition. Exposure to 5 Hz, 2.0 $m.s^2$ whole-body vibration was found to produce back muscle fatigue earlier and to a greater extent than quiet sitting (Hansson, Magnusson, and Broman, 1991; Magnusson, Hansson, and Broman, 1988). Fatigue in this study was assessed using mean frequency of EMG at L3 and T7 levels while subjects supported 4 kg at chest level and bent forward at 20 degrees (Hansson, Magnusson, and Broman, 1991; Magnusson, Hansson, and Broman, 1988).

Conflicting research (Pope, Wilder, and Donnermeyer, 1986; Wilder et al. 1984) reports that a two hour static sitting condition produced greater subjective (visual analog scale) and objective (EMG center frequency shift) muscle fatigue than two

hour exposure to a vibrational sitting condition designed to reproduce the UH-1H helicopter vibration profile. This indicates that vibration, *per se*, may not be the sole etiological factor in paraspinal muscle fatigue, since static sitting also produces significant muscle fatigue and associated back pain.

Muscle fatigue normally associated with awkward reaching and overhead arm posture is accentuated by hand-arm vibration. This occurs in the primary mover muscles, muscles with increased length, and muscles with increased stiffness due to contraction (Rohmert et al. 1989). Posture may, therefore, be a factor in predisposing muscles to vibration-induced fatigue, since posture will contribute to the determination of muscle length and function.

The sitting posture deemed to produce the least myoelectric activity in the thoracic and lumbar regions of the back is a position of 120 degrees backrest inclination, 5 cm of lumbar support, and 13.5 to 18.5 degrees of seat inclination (Hosea et al. 1986; Andersson et al. 1974), with backrest inclination the most important parameter (Andersson, Jonsson, and Ortengren, 1974). Unsupported sitting produces greater disc pressure and myoelectric activity than standing or supported sitting (Andersson et al. 1975). These factors must be controlled in any analysis of the effects of seated posture or seated exposure to vibration and impacts.

Muscle activity has been shown to demonstrate phasic bursts of activity separated by silent periods in response to low frequency (less than 12 Hz) vibration (Robertson and Griffin, 1989; Cursiter and Harding, 1974). This phasic response is unchanged or slightly diminished in magnitude during prolonged exposure (2.5 hours) to whole-body vibration, while tonic muscle activity increases (Robertson, 1986; Robertson and Griffin, 1989). The apparently dichotomous effects on tonic and phasic muscle activity have been referred to as the "vibration paradox" (Desmedt and Godaux, 1978). The phasic response becomes less significant in magnitude, relative to the background muscle activity, and may indicate a diminishing responsiveness of the musculature as exposure duration is increased. The elevated tonic activity during vibration exposure can be seen as a protective, stabilizing response. However, the progressive increase in magnitude of EMG response to a constant level stimulus is also indicative of muscle fatigue. The developing fatigue and the decreased phasic responsiveness of muscle may reduce the body's ability to adequately respond to additional perturbations, such as impacts or jolts.

The rate of recovery from muscle fatigue has been used by several authors to assess the state of the muscle system after exercise or isometric test contractions (Kuorinka, 1988; Roy et al. 1990; Zwarts, Van Weerden, and Haenen, 1987; Petrofsky, 1981; Miller et al. 1987; Mills, 1982); however, no data regarding recovery from vibration-induced fatigue were reported. A healthy

system will recover more quickly upon cessation of vibration (Roy et al. 1990). Kuorinka (1988) found the rate of restitution of EMG spectral parameters to depend on the level of fatigue achieved. For both large and small loads, recovery from isometric contractions to complete volitional fatigue was found to occur within 5 minutes. Submaximal fatigue resulted in faster recovery rates. Literature cited by Kuorinka (1988) report complete restitution of the EMG spectrum to take anywhere from 1 minute to several hours, depending upon the experimental conditions and method of measurement. Quantification of recovery rates may provide a method of assessing both the severity of single exposures and the cumulative effect of sequential vibration exposures. More severe single exposures or any cumulative effect from sequential vibration or impact exposure would be expected to induce a greater level of fatigue and a slower rate of recovery from fatigue. Moussavi et al. (1989) found that spectral restitution occurred before force generating capacity was restored after volitional fatigue using relatively low loads (30 percent maximal voluntary contraction). This suggests that measures of force generating capacity or force-EMG relationships may be a useful complement to spectral restitution in assessing recovery from fatigue.

Timing of the Muscle Response to Vibration

Muscle response to whole-body vibration involves complex central and peripheral neural feedback mechanisms combined with the inherent viscoelastic properties of the musculoskeletal system (Seidel, Bluethner, and Hinz, 1986; Seidel, 1988) and an electromechanical delay between depolarization of the muscle cell and the generation of twitch tension (De Luca, 1985). The properties and interactions of these systems will determine the timing and efficiency of the muscle response. Muscle response under different conditions of vibration has been utilized to gain an understanding of various sensory control mechanisms, including muscle spindle, vestibular, and visual modalities of postural control (Robertson and Griffin, 1989; Desmedt and Godaux, 1978; Matthews and Watson, 1981; Burke et al. 1976). In addition, the timing of the muscle response holds significance in the determination of forces at the spine, since changes in muscle tension contribute to compression and torque (Hagena et al. 1986; Seroussi, Wilder, and Pope, 1989; Seroussi et al. 1987; Ortengren, Andersson, and Nachemson, 1981).

The relationship between muscle response and a perturbing force is characterized by three parameters: the magnitude of the phasic response, the temporal lead or lag, and the phase relation. The timing of the muscle response relative to acceleration has been studied using a direct comparison of maximum and minimum peak occurrences (Seidel, 1988; Seidel, Bluethner, and Hinz, 1986; Seidel and Pietschmann, 1979). The phase relationship between average acceleration and average EMG

activity during exposure to sinusoidal vibration is determined using the cross-correlation of these signals (Robertson, 1987; Robertson and Griffin, 1989). Phase relationships are more difficult to obtain for random vibration exposures; however, a transfer function method using cross spectral density between acceleration and rms EMG has been used (Robertson and Griffin, 1989). The comparison of EMG maxima and minima to acceleration profiles requires the incorporation of an electromechanical delay to account for the time between depolarization of the muscle and tension generation (Robertson, 1986). The electromechanical delay for human paraspinal muscles is typically assumed to be about 50 ms. However, this does not account for dynamic changes in the time required to produce tension, which are a function of muscle length, prior contractile status, and whether an eccentric or concentric contraction results.

The timing and relative magnitude of the muscle response is of interest in determining whether there is a change in muscle length. Lengthening or shortening of a muscle occurs only if there is a difference of magnitude between an externally applied force and the force developed by the muscle group involved (Berthoz and Metral, 1970). This is of significance when considering the maintenance of postural stability and limb position during exposure to an oscillating force. Berthoz and Metral (1970) found that rapidly applying and removing a load to the forearm produced an oscillating EMG in biceps brachii and brachioradialis. At frequencies above 5 Hz, the EMG activity from the previous burst was still present when the next oscillation began. The authors related this to the relaxation time of electrically stimulated muscle (250 to 300 ms) which is considerably longer than the period of oscillation at these frequencies (Berthoz and Metral, 1970). This would suggest that the consecutive burst of EMG activity was occurring while the muscle was still under tension. It was also found that rapid loading and unloading at 200 ms or less induced an overshoot in flexion upon unloading. The overshoot was deemed to be caused by a combination of flexion in response to loading and simultaneous flexion due to removal of the load a short time later. This early study serves to illustrate the importance of a timely and appropriate muscle response in order to maintain a stable posture. Poorly timed responses to consecutive loading and unloading, as in vibration or impacts, may lead to an exaggerated muscle response compared to the loading phase alone.

An untimely muscle response to low frequency sinusoidal whole-body vibration was first reported by Seidel and Pietschmann (1979), who demonstrated a clear time differential at 2, 4, and 8 Hz. They noted minimal EMG activity during peak $+G_Z$ acceleration at these frequencies. Later work by Seidel, Bluethner, and Hinz (1986) found that the timing difference was minimal at 4 to 4.5 Hz, which they suggest may contribute to resonant properties of the system since acceleration-synchronous muscle activity would increase stiffness and decrease damping properties of the muscles and connected structures. Seidel (1988) found the timing of the

muscle response to low frequency vibration was well synchronized from 0.315 Hz to 1.25 Hz, but changed from a phase lead at 2.5 Hz to a phase lag at 5.0 Hz. Robertson (1987) found similar results with muscle activity showing no phase difference over seat acceleration and force at 0.5 Hz; a phase lead from 1 Hz to 4 Hz (maximum lead of 117 degrees at 2 Hz); but reversing to a phase lag at higher frequencies (117 degrees at 8 Hz, 396 degrees at 16 Hz). Robertson and Griffin (1989) confirmed this trend for sinusoidal vibration and demonstrated similar phase shifts for random whole-body vibration with 0.25 to 8 Hz components. The shifting phase relation was interpreted to indicate a change in the predominant control mechanism from visual and vestibular to proprioceptive and mechanoreceptor modalities.

Visual and vestibular cues are deemed to predominate sensorimotor control at frequencies below 2 Hz in what Seidel (1988) called a "conservative mode" of motor control, which can be voluntarily over-ridden. Robertson and Griffin (1989) were unable to experimentally alter the phase or magnitude of muscle response to vertical and fore-aft vibration at 1 to 16 Hz using visual field stabilization (visual field moved with the subject) or vestibular stimulation (head rotation). They attributed this failure to rapid sensory compensation and adaptation. Proprioceptive and mechanoreceptive control are deemed to become more significant above 2 Hz (Robertson, 1987; Seidel, 1988). Seidel (1988) believed that the poor timing of muscle response at frequencies between 2 and 16 Hz may have been caused by sensory conflict, while Robertson and Griffin (1989) postulated an integration of peripheral and central mechanisms. Stretch reflex was established as a contributor to the muscle response at low frequency, but not the sole mechanism, since one subject with a proprioceptive sensory neuropathy was shown to have a relatively normal phasic muscle response to low frequency vibration. However, the phase lag was greater and the magnitude of response between 4 and 8 Hz was below normal (Robertson and Griffin, 1989).

In determining phase relationships between two continuous signals, it is necessary to be able to pair the two signals in time such that differences in phase can be attributed to lead or lag. Very few authors have identified the method employed to pair their signals. Seroussi et al. (1989) used the time moment of the crosscorrelation for one full period of oscillation to determine the phase relationship between seat acceleration and peak torque. They report an average time delay of 81 +/- 19 ms across all frequencies tested.

Muscle Activity and Forces at the Spine

The timing and magnitude of muscle response has been reported to be a primary mechanism determining prediction of load on the lumbar spine. Biomechanical models of dynamic spinal loading are

inadequate if they ignore the muscle response or assume optimal muscle function and timing (Seidel, Bluethner, and Hinz, 1986). Seidel (1988) suggests the use of a time-variant spring characteristic in which contraction has high stiffness and relaxation has low stiffness to account for the dynamic muscle response. Seroussi, Wilder, and Pope (1989) demonstrated that predictions of torque underestimated actual torque production during seated whole-body vibration when the dynamic muscle response was not incorporated into the model. It was further found that the average and peak torque imposed on the spine during an isometric horizontal pull from a seated position were significantly greater during whole-body vibration than during static sitting (Seroussi, Wilder, and Pope, 1989; Seroussi et al. 1987). This may be due to the added torque imposed by seated acceleration, but may also be a result of the added tonic muscle activity that accompanies whole-body vibration.

The dynamic nature of the vibrating environment produces some problems for calibration and normalization of EMG. An EMG-force calibration must be performed to estimate tension produced by the muscle, and therefore to estimate compressive load or torque on the spine. In a static situation, a series of isometric contractions of known force can be used to calibrate the EMG-force relationship. This calibration is only valid for a given joint angle or muscle length across the tension range of the test contractions (Mirka, 1991; Mirka and Marras, 1991). According to the length-tension relationship, the tension generating capacity of muscle is dependent on muscle length; therefore, the relationship between maximal voluntary contraction (MVC) and any lesser tension will change with muscle length. In the dynamic situation, a greater magnitude of EMG is associated with a given muscle tension (Westgaard and Bjorklund, 1987; Mirka and Marras, 1991). This is due to changes in muscle length and changes in the portion of muscle within range of surface electrodes as the muscle contracts and the joint angle changes (Mirka, 1991). The use of a single reference contraction for normalization of dynamic erector spinae EMG has been found to produce errors of greater than 75 percent (Mirka, 1991; Mirka and Marras, 1991). Whole-body vibration results in phasic contraction of the paraspinal musculature, which will require either a complex force-length-EMG calibration or some estimate of the average EMG-force relationship based upon the range of muscle lengths anticipated. Mirka (1991) used a complex calibration procedure to establish a force-EMG relation for the erector spinae muscles using a KIN-COM dynamometer to control force and trunk angle. EMG was recorded from L3 level for MVC at ten different trunk angles as well as during dynamic movement with a preset angular velocity; however, it is unclear how this was applied to the EMG signal. EMG normalization methods must be validated through the entire range of motion for the muscle of interest (Oberg, Sandsjo, and Kadefors, 1991; Mirka and Marras, 1991).

Normalization of integrated EMG (IEMG) data for a group of subjects or trials has been successfully accomplished for isometric contractions using the following formula:

Norm EMG = (EMG - Min EMG) / (Max EMG - Min EMG),

where Norm EMG is the normalized IEMG, EMG is the measured IEMG, Min and Max EMG are the minimum and maximum IEMG values over all conditions (Mirka, 1991; Mirka and Marras, 1991). This method allows for analysis of group data, elimination of inter-individual differences, and elimination of direct current (DC) bias.

Muscle Response to Impacts

Very little research has reported the dynamic response of the musculature to mechanical shocks or impacts. Robertson and Griffin (1989) monitored EMG of paraspinal muscles during impact accelerations superimposed on sinusoidal vibration. They produced graphical representations of EMG amplitude to demonstrate a phasic response that closely resembles the profile of the acceleration; however, no quantification of timing, phase, or recovery time was performed. Tennyson et al. (1977) studied the timing of the paraspinal muscle response relative to 30 and 50 $m.s^{-2}$ $+G_z$ impacts in beagles, and found a delay of 21 to 58 msec between the onset of impact and the onset of muscle response, and a delay of 45 to 135 msec between the peak impact acceleration and the peak EMG activity. Seroussi et al. (1989) found a comparable delay of 72 to 100 ms between seat acceleration and peak torque production by the paraspinal musculature at L5 in human subjects. This delay is less than that typical of the human stretch reflex; nevertheless, it was deemed by both authors that this delay was sufficiently long to negate any beneficial effect from the increase in muscle tension. The generation of tension by a muscle occurs after the transmission of impact through spinal tissue.

The use of EMG recovery holds promise in quantifying the effect of successive impacts on the musculature. If one assumes that the muscle response to the background vibration reaches a pseudo-steady state, the rate of return to that steady state after impact may be an indication of the ability of the muscle to cope with additional perturbations. The response of single perturbations on oscillating systems has been used to gain information about the behaviour of other complex biological systems (Glass and Mackey, 1988). Phase space trajectories have been used to characterize noisy, apparently random physiological processes, such as heart rate, in normal and disease states (Goldberger, Rigney, and West, 1990). The use of phase-space trajectories to quantify the steady state, the shift from steady state, and the return to steady state may be of use in determining the plasticity of the muscle response to successive impacts superimposed on whole-body vibration.

Conclusions

Electromyography of paraspinal muscles has a multi-faceted role in the development of a health hazard index for exposure to whole-body vibration and repeated impact. It should be noted that the use of paraspinal EMG, in general, requires control of postural conditions and the effects of prolonged static sitting. Normalization procedures will require validation for the experimental conditions used in the USAARL tests. The following outlines both the utility and the limitations of EMG with respect to health hazard assessment.

The contribution of muscle tension to forces on the spine can be estimated using EMG; however, the precision of this estimate is limited by the difficulty in adequately calibrating an EMG-force relationship for a dynamic process, and by the complex phase relationship between acceleration and muscle response.

The relative timing of tension generation by muscle is important for an effective homeostatic response to vibration and impacts; however, the determination of a phase differential between acceleration and tension generation requires the incorporation of an electromechanical delay between onset of myoelectric activity and tension production. This delay is influenced by the dynamic characteristics of the muscle, and can only be roughly estimated.

Fatigue of paraspinal muscles can be quantified through various analytical techniques; however, the significance of any measured fatigue can only be inferred with respect to back pain and a diminished capacity for postural control.

The use of post-vibration or post-impact recovery rate of EMG from a fatigued condition may provide an indication of relative severity of exposure; however, no inference of tissue damage can be drawn directly from EMG. Complementary analysis of biochemical markers for tissue damage may allow the establishment of an association between EMG parameters and tissue damage.

The ability of the musculature to respond to successive impacts can be estimated using a recovery rate model; however, the method of defining the steady state response to background whole-body vibration has not been established. The use of phase space trajectories may provide a method of quantifying steady state recovery.

Biodynamic Response: Transmission, Impedance and Apparent mass

Introduction

When the human body is subject to disturbance, as in exposure to vibration or impact, it demonstrates a dynamic response. The displacement of tissues and the forces transmitted by them alters as a function of time. The precise nature of the response is determined by the mass, stiffness and damping of each tissue. External displacements applied to the body may be amplified or attenuated by different tissues or physiological systems, resulting in local stresses within the body. A useful method of assessing the potentially harmful effects of vibration and impacts is to measure the relative displacements and hence stresses of different regions of the body in response to the applied displacement (or acceleration), amplitude and frequency. Transmission of acceleration can then be expressed in terms of a transfer function. This defines the relative magnitude and phase relationship of the output acceleration at a particular region (for example, at the head) compared with the input acceleration (for example, at the seat). Knowledge of the acceleration transfer function provides insight into the behaviour of the various body sub-systems, and enables the investigator to assess at what input acceleration levels and frequencies a particular tissue is more likely to suffer damage.

Transmission

A substantial body of knowledge has been reported concerning the transmission of vibration to body segments. Data exists for standing, supine and seated postures, including seated erect, relaxed and with or without a back rest (Magid and Coermann, 1963; Dupuis and Zerlett, 1984; Rowlands, 1977; Rao and Jones, 1975). The extent of the literature available reflects the ease with which transmission measures can be obtained from certain body subsystems, particularly seat to head transmission. In contrast, data on the vibration response of internal systems such as the abdominal or thoracic tissues are relatively sparse. Transmission can be reported in either graphical or mathematical form as a transfer function representing the ratio of acceleration (or displacement) of a body segment (output a_o) to that of the seat or floor (input a_i) as a function of frequency (f_i). Thus:

$$a_o : a_i = G(f_i)$$

Transmission from the seat to the head

A number of authors have reported seat to head transmission in the z-axis (Muller, 1939; Magid and Coermann, 1960; Dupuis, 1969; Paddan and Griffin, 1988). In general, results show transmission to approximate unity at frequencies below 2 Hz. A primary resonance occurs between 4 Hz (Magid and Coermann, 1960; Dupuis and Zerlett, 1984; Rowlands, 1977) and 6 Hz (Pope et al. 1985; Paddan and Griffin, 1988) with a maximum seat to head transfer factor of 1.5 to 2.5. The seat to head transfer function may also exhibit one or two additional resonances at 9 to 12 Hz and 12 to 16 Hz (Magid and Coermann, 1960; Wilder et al. 1982; Paddan and Griffin, 1988) with maxima of 0.5 to 1.5. The exact nature of the transfer function has been shown to vary in response to posture and seat back support. When sitting relaxed with no backrest, Magid and Coermann (1960) reported a single resonance at 4 Hz. The transfer function decreased rapidly from a maximum of 2.2 to values less than 1.0 above 6 Hz. An erect posture, and back support tends to shift the primary resonance to a slightly higher frequency with a lower peak value, whereas the transfer factors of the two higher resonances are increased (Magid and Coermann, 1960; Rowlands, 1977; Paddan and Griffin, 1988).

An input acceleration in the z direction at the seat also invoked head motion in the x-axis (fore-aft translation), and y-axis rotation (pitch) (Paddan and Griffin, 1988). Transmissibility in the x-axis (x head/z seat) was greater with a back support having a peak transfer factor of 1.0 to 2.0 at 6 Hz, compared with 0.5 to 1.0 without back support. The seat to head transfer functions were also found to be dependent on pelvic angle (Wilder et al. 1982; Messenger and Griffin, 1989) and head angle (Cooper, 1986). As pelvic angle was reduced from 105^{o} to 85^{o}, the transfer factor was increased at frequencies above 6 Hz (Messenger, 1987).

Paddan and Griffin (1988) found that a back support increased the seat to head transmission of horizontal seat vibration. This factor must be considered in seat design, especially if a back support is introduced to relieve spinal loading. Seat acceleration in the x-axis produced significant output accelerations at the head in the x-axis, z-axis and y-axis rotation (pitch), with resonances at 2 Hz and 6 to 10 Hz and transfer factors of between 0.5 and 2.0. With no back support, the second resonance was eliminated. Seat acceleration in the y-axis produced predominantly y-axis motion at the head with a single resonance at 2 Hz and peak transfer factor of approximately 1.5. There was little motion of the head above 8 Hz.

These results broadly agree with the data of Paddan (1986) obtained from a tracked armoured fighting vehicle driven over a rough cross country course. However, whereas Paddan (1986) found z-axis seat acceleration to be the main source of z-axis, x-axis and pitch motion, the laboratory study of Paddan and Griffin

(1988) suggests that x-axis seat acceleration also has a major effect on head motion in these three directions, particularly when the back is supported.

Thoracic Abdominal Displacement

Knowledge of the dynamic response of the thorax and abdominal regions is relatively sparse compared with that of the head and spinal column. Coermann et al. (1960) studied the dynamic properties of the thorax-abdomen system using measures of abdominal acceleration, thoracic expansion, and respiratory air flow. Subjects were vibrated in a supine posture, with the accelerations applied in the horizontal plane (biodynamic z-axis vibration). The maxima of all three transmission functions coincided at 3 to 4 Hz, suggesting a close coupling between the abdominal mass, lungs and chest wall (Coermann et al. 1960). Transmission amplitude fell rapidly at frequencies above 5 Hz, although there was some evidence of a secondary resonance at 6 Hz. Lange and Edwards (1970) used circumferential strain gauges to measure thoracic and abdominal displacement of supine subjects lying on a shake table during sinusoidal vibration in the vertical direction (biodynamic x-axis). Resonant frequencies were higher (5 to 7.5 Hz) than in the z-axis (Coermann et al. 1960), and decrease of transmission above 8 Hz was less rapid. The authors also reported that tensing of muscles tended to increase resonant frequency by approximately 1 Hz, and that the effect of input acceleration level on resonance frequency and transmission amplitude was non-linear. Increases of input acceleration amplitude from 2.0 to 5.0 $m.s^{-2}$ produced a reduction of resonance frequency from 7.5 Hz to 5 Hz. The shift of resonance frequency is similar to the findings of Fairley and Griffin (1989) when measuring apparent mass of seated subjects.

Dupuis and Zerlett (1984) report the linear displacement of the abdomen and sternum of 14 supine subjects. Results showed agreement with the above authors, with resonance frequencies occurring at 2 to 4 Hz in the (biodynamic) z-axis, and 8 Hz in the x-axis. Transmission was greatest in the y-axis with maxima of 3.0 to 4.0 at a resonance frequency of 1.5 Hz As subjects were vibrated while in a supine posture in the above studies, the data does not necessarily reflect the mechanical properties or resonance frequencies of the thorax and abdomen when seated in an erect posture.

Sandover (1982) reported pressure response of the lower intestine of upright seated subjects to z-axis acceleration. The response function was similar to that of apparent mass, having a peak pressure at 5 to 6 Hz.

Donati and Bonthoux (1983) reported a seat to thorax transfer function in the z-axis, using an accelerometer strapped to the chest at the sternum. A single degree of freedom was observed

for the thorax, with a resonance frequency of 4 Hz, and a maximum transfer factor of approximately 2.3. The resonance of the thorax coincided with the primary resonance of the driving impedance at the seat.

Accelerations Of The Spine

Low back pain and chronic degeneration of the spine have been associated with exposure to whole-body vibration, and repeated impact (Dupuis and Zerlett, 1987; Hansson and Holm, 1991; Sandover, 1982; Hulshof and van Zanten, 1987). Epidemiological studies suggest that the lumbar spine is more susceptible to damage and discomfort in response to vibration and repeated impact than the thoracic and cervical regions, whereas the thoracic spine may be more susceptible to damage in response to a single impact of higher magnitude (Jones, Madden, and Luedeman, 1964).

Numerous researchers have investigated the transmission of accelerations from the seat to different levels within the spinal column. Non-invasive measures have been made using miniature accelerometers attached to the skin over the spinal process (Zagorski et al. 1976; Seidell, Bluethner, and Hinz, 1986). Invasive measures of spinal accelerations have also been made in a small number of subjects by inserting a pin into the spinal process and attaching to it miniature accelerometers (Hagena et al. 1985; Andersson et al. 1985). Invasive measures have shown that a z-axis acceleration at the seat results in z-axis and x-axis accelerations, and pitch (y-axis rotation) at the vertebra (Panjabi et al. 1986; Sandover and Dupuis, 1987; Pope et al. 1991). Panjabi et al. (1986) measured accelerations of the L1 and L3 vertebrae and sacrum at an input level of 1.0 and 3.0 $m.s^{-2}$ at the seat. A resonant frequency of 4.4 Hz and transmission factor of 1.6 (mean) was measured in the z-axis at both L1 and L3. The resonant frequency at the sacrum was higher at 4.8 Hz and the transmission greater (mean = 1.9). Accelerations in the x-axis displayed transmission factors of 0.2 to 0.8, but no obvious resonant frequency. The resonant frequency of pitch could not be determined from the data. Transmission factors were not significantly different at 1.0 and 3.0 $m.s^{-2}$. In an earlier publication by the same group, a pitch resonant frequency of around 5 Hz is reported (Andersson et al. 1985).

Sandover and Dupius (1987) reanalysed cinematographic data of spinal motion gathered by Christ and Dupuis (1966) from pins driven into the processes of T12, L1, L2, L3 and L4. Data was collected at 2 to 7 Hz sinusoidal seat motion of amplitude 10 mm peak to peak. Results demonstrated resonant behaviour at 4 Hz in the z-axis, x-axis and pitch (y-axis rotation) directions. It was estimated that rotation of the vertebrae (flexion) caused a 24% to 45% overestimation of z-axis displacement of the L4

vertebra centroid when measured at the target (pin), or a 20% error if measured at the skin surface. At vibration levels of 1.0 $m.s^{-2}$ a y-axis rotation of 1^{o} peak to peak existed between adjacent vertebra. The authors emphasized the importance of spinal flexion in obtaining accurate data of z-axis accelerations of the vertebra. The relative displacement of vertebra in the z-axis was small, with a maximum value at 4 Hz of 2 mm between L2 and L4. This is compared with the absolute displacements of the vertebrae of approximately 20 mm, yielding a seat to spine transmission factor of 2.0. The bending was complex and involved phase differences along the spine with a possible rocking motion of the pelvis.

Pope et al. (1991) attached a strain gauge extensometer linkage system to pins inserted to the spinous process of adjacent vertebrae. Seated subjects were vibrated in the z-axis at 0.4 to 1.7 $m.s^{-2}$ at 5 Hz and 8 Hz. The system measured relative motion between the vertebrae of 0.1 to 1.1 mm in the z-axis, 0 to 0.4 mm in the x-axis and 0 to 0.4 degrees of pitch. Although considerable variance in response was exhibited between subjects, the results tend to support the findings of Sandover and Dupuis (1987).

Pope et al. (1986) compared displacements at the skin surface with those measured from pins inserted in the L3 spinous process and the posterior superior iliac spine. Results of the two methods were substantially different showing relative peak displacements of 0.2 to 0.5 $mm/m.s^{-2}$. Although absolute values of displacement were not provided, results suggest skin surface measures to be unreliable, with substantial error due to artefact.

To avoid invasive techniques, Hinz et al. (1988) developed a method of calculating bone accelerations from miniature accelerometers attached to the skin. The soft tissue between the spinous process at L3 and T5 and the accelerometer were modelled as a Kelvin element, whose parameters describe an approximate transfer function between the bone (input) and skin surface (output). Parameters were determined experimentally from free damped oscillations of the accelerometer - skin complex in the z-axis. The prediction of the model was that bone accelerations are smaller. The rms acceleration ratios of skin to bone were 1.2 to 1.4 at 1.5 Hz and 1.5 to 2.0 at 8 Hz. Similar findings are reported by Smeathers (1989). Although this technique provides an improved accuracy for non invasive measures of spinal acceleration, it is restricted to axial motion and does not correct for error due to pitch caused by flexion-extension of the spine (Sandover and Dupuis, 1987; Hinz et al. 1988). Hence measures of relative acceleration in the z-axis from skin surface accelerometers cannot be used to derive disc compression without further correction for flexion-extension of the spine. The results of the above studies of spinal motion confirm complex internal loads with coupled bending, compression and shear forces (Hinz et al. 1988).

Vibration And Impacts in Normal Activity

The human body is subjected to impact and vibration during the course of normal activities such as walking and running. Thus, the investigation of segmental accelerations in such activities can provide useful information in the assessment of acceptable levels of exposure to repeated impact and shock in less familiar conditions.

Light, McLellan and Klenerman (1980) reported peak accelerations when walking of 50 $m.s^{-2}$ and 5.0 $m.s^{-2}$ at the tibia and skull respectively, indicating an attenuation of impact of 0.1 between the tibia and head. Accelerations were halved when hard heeled shoes were replaced by resilient heeled shoes. The authors discuss the hypothesis that these acceleration transients may contribute to osteoarthritic degeneration, and that shear stresses in para-osteal tissue may aggravate low back pain. Rao and Jones (1975) measured mean peak to peak accelerations of 5.8 to 8.4 $m.s^{-2}$ at the head and shoulders when walking. Resonant frequencies of 5 Hz at the shoulders and head, and 19 Hz at the head were detected. Corresponding rms accelerations averaged 2.2 $m.s^{-2}$ at 1.45 Hz (step frequency), 1.1 $m.s^{-2}$ at 5 Hz and 0.2 $m.s^{-2}$ at 19 Hz. Comparison with Fatigue Decreased Proficiency Limits of ISO 2631 indicated accelerations fell within the 25 to 60 min. curves at 1.45 Hz and 1 to 2.5 hr. curves at 5 Hz. However, these accelerations are not directly comparable with accelerations measured at the seat pan as in ISO guidelines. Normally there would be a transfer function between the seatpan and head. Data in the literature suggest the acceleration at the head is approximately 1.5 times input acceleration at 5 Hz, when seated erect or standing. Thus, allowing for a transmission factor of 1.5 and comparing the head/shoulder accelerations of Rao and Jones (1975) with the ISO Exposure Limit would predict a safe exposure of approximately 8 hours (input acceleration = 0.63 $m.s^{-2}$) for normal walking.

Capozzo (1982) assessed upper body vibrations at walking speeds of 1.0 to 2.4 $m.s^{-1}$. Acceleration measured at the pelvis, shoulders and head were frequency weighted in accordance with ISO 2631 and compared to exposure limits. Z-axis accelerations were correlated with walking speed, having a safe exposure limit of 4 hours at 1.2 $m.s^{-1}$ (1.0 $m.s^{-2}$ rms), reducing to 16 mins at 2.3 $m.s^{-1}$ (5.2 mph; 4.3 $m.s^{-2}$ rms). The authors point out that, while these speeds are not considered to be damaging, most individuals would be constrained to these exposure times by onset of fatigue. The data does not take account of transmission factors when comparing accelerations of body segments to the ISO 2631 standard. Nevertheless, the above papers provide valuable data in determining levels of repeated impact, rms accelerations, and exposure durations which are known to be safe and free of long term health effects.

Simic (1977) measured accelerations at the pelvis and head for step frequencies of 1.6 Hz to 3.26 Hz. For normal walking,

accelerations in the z : x : y directions were in the ratio of 1 : 0.8 : 0.5 at the pelvis. z-axis acceleration increased from 3.7 $m.s^{-2}$ for slow walking (1.6 Hz step) to 16.5 $m.s^{-2}$ (3.1 Hz step) when running.

The question of whether impacts experienced in walking and running are directly comparable to those experienced when seated is discussed by Sandover (1979). Head accelerations were compared between externally applied vibration (seated and standing) and ambulation (walking and running). Input-head acceleration transmission factors when seated or standing were 0.9 to 1.7, a much lower factor than measured by Light, McLellan and Klenerman (1980) for walking. Pelvic accelerations were at least as great as those of the head during walking and running. Results indicated that the body moves with a constant displacement amplitude (of 75 mm) and that acceleration amplitude increases with the square of gait frequency. Sandover found a lower sensitivity to ambulatory accelerations. He suggests that muscle activity and posture in locomotion may act to attenuate movements in sensitive areas of the body (e.g. the viscera). This suggestion is supported by Smeathers (1989) who found that the dominant resonance frequency of 5 Hz displayed in response to externally applied vibrations was not reproduced in their analysis of spinal accelerations in walking and running. Sandover also notes that differences in sensitivity between walking and externally applied vibration may be psychological rather than physiological.

Impedance

The behavior of the body in response to an input vibration may also be represented by the mechanical impedance of the system. Mechanical impedance is analogous to electrical impedance, where force, velocity and displacement are equivalent to voltage, current and charge respectively. The system properties of mass, compliance and damping are likewise analogous to inductance, capacitance and resistance of an electrical circuit. The measurement of mechanical impedance is of value in modelling the various subsystems of the body in that it allows the researcher to assign absolute values to the system parameters.

Impedance is defined as the ratio of force to velocity. In an oscillating system the force and velocity may not be in phase with respect to one another. Hence impedance is expressed as a complex number. In human body dynamics, system impedance can be measured either as "driving point impedance", where the force and velocity are measured at the same point (for example the seat), or as "transfer impedance", where the force is measured at one point (for example the seat) and the resultant velocity is measured at another (for example the head or spine). Measures of transfer impedance can provide further insight into the mechanical properties of a particular subsystem in the same

manner as nodal analysis within an electrical circuit. In addition, knowledge of impedance obtained under conditions of steady state sinusoidal displacement can be used to predict the response of the body to impact forces applied to the seat.

Coermann et al. (1960) measured the driving impedance of 5 subjects when seated. At frequencies below 2 Hz, the body behaved as a pure mass. The impedance response exhibited resonance peaks at 5 Hz and 11 Hz The authors attributed resonance at 5 Hz to the bending elasticity of the spine and pelvis. The resonance at 11 Hz was considered to be an additional resonance of the pelvis. Values of mechanical impedance were found to vary in response to posture.

Donati and Bonthoux (1983) also measured mechanical driving impedance between 1 and 10 Hz. Results were similar in form to those of Coermann et al. (1960) and also to the ISO Standard 5982, with the exception that the two resonances occurred at slightly lower frequencies (4 Hz and 8 Hz) and impedance was of lower magnitude than the ISO 5982. The authors attribute the difference to posture and mass, as their subjects had the hands and feet supported independently (but moving with the seat). A large variance existed among subjects; whereas resonance at 4 Hz was well marked, for other resonant frequencies the predominance of damping precluded this phenomenon.

Apparent Mass

The characteristics of vibration response have also been described in the form of apparent mass, representing the complex ratio between applied force and acceleration. Selection of apparent mass as a method of displaying system response results in an output function which is more readily interpreted by the experimenter, and which can be normalized to the body weight of each subject. At zero frequency, the apparent mass in the z direction simply represents the body mass of the subject (Fairley and Griffin, 1989).

To overcome the problems experienced in comparing the driving point impedance functions of different authors using a variety of units, Sandover (1982) converted the z-axis impedance data of several investigators to apparent mass. Results show a flat response at low frequencies, a resonance peak at 4 to 6 Hz, and a rapid attenuation of the apparent mass function at higher frequencies. The secondary resonance displayed in the driving point impedance data is less obvious in the apparent mass function. Fairly and Griffin (1989; 1990)) measured apparent mass in different postures and at different levels of acceleration in the x, y and z axes. An important finding was that the resonance frequency of the apparent mass function in the z direction decreased with increasing acceleration magnitude (between 0.25 and 2.0 $m.s^{-2}$), whereas resonance frequency

increased in response to muscle tension, and from slouched to erect posture. This finding contradicts the expectation that muscle tension and hence resonance frequency would increase with acceleration magnitude, and suggests that dynamic response of the body is non-linear.

Acceleration of the seat in the x direction (fore-aft) produced a heavily damped response, with resonances of apparent mass at 0.7 Hz and 2.5 Hz. When provided with a backrest, a single more pronounced resonance occurred at approximately 3.5 Hz. The authors suggest that the lower resonance represents body sway, while the secondary resonance represents linear translation rather than rotation of the torso. It was observed that without a backrest, body sway was controlled by voluntary or involuntary muscle contraction (Fairley and Griffin, 1990). Lateral motion produced a less well defined response of apparent mass, with resonances of 0.7 Hz and 2 Hz without back support, and a single resonance at approximately 1.5 Hz when supported by a backrest. The effect of the backrest was much less pronounced in the lateral direction.

Conclusions

Despite the wealth of information on transmission, impedance and apparent mass, the dynamic response of individual body segments to vibration and impact remains imperfectly defined. The uncertainty arises due to the strong coupling action of adjacent body structures demonstrated in experimental results, confounding the assignment of mechanical properties to specific structures.

A resonance frequency of 5 Hz has been attributed to the spinal column and pelvis (Sandover, 1982). However, the intervertebral discs have been shown to be too stiff in axial compression to attenuate low frequency impacts (Markolf, 1970; Belytschko and Privitzer, 1978; Smeathers, 1989). Thus, the resonance frequency of 5 Hz attributed to the spine probably derives from the combined properties of upper torso mass, flexibility of the spinal column, and the stiffness and damping of the supporting muscles and ligaments. Belytschko and Privitzer (1978) concluded that the resonance of driving point impedance shown at 5 Hz resulted from a combination of pelvic, visceral and spinal elements, and reflected the elastic properties of the buttocks, abdominal wall and spinal flexion respectively.

Although measures of driving point impedance and apparent mass provide useful indications of the response of the body, these measures do not provide sufficient detail to determine the behaviour of, or stresses acting on, individual systems such as the abdomen or spine. In particular, there is a lack of information regarding non-linearities of individual systems, particularly in response to impacts.

Biomechanics

Introduction

Whole-body vibration and repeated shock cause mechanical disturbances of the body. Epidemiological studies point towards low back pain and injury due to accelerated degeneration of the spinal unit as a hazard of chronic vibration expsoure, whereas impact injuries involve fractures of the vertebrae. Mechanical systems fracture under severe loading and suffer fatigue failure in response to low level vibration. Hence, there is an obvious correlation between the effects of mechanical shock and vibration on living systems and the failure modes of engineering materials. It follows that any attempt to gain a quantitative understanding of the physical and mechanical processes underlying the adverse effects of vibration must begin with an investigation of the mechanical properties of tissue.

Material Properties of the Spine

The biomechanical properties of the spine have been studied extensively in vitro (Kazarian, 1972; Kazarian, 1975; Markolf, 1970; Wilder, Pope, and Frymoyer, 1987; Wilder et al. 1982; Cyron and Hutton, 1978; Tencer, Ahmed, and Burke, 1982) and to a lesser extent in vivo (Nachemson and Morris, 1964; Andersson, 1980; Corlett et al. 1987; Christ and Dupuis, 1966; Pope et al. 1991). Study of material properties in vitro has included the behaviour of the isolated spinal unit, comprising an intervertebral disc and the superior and inferior vertebrae. Studies include the load deflection characteristics under conditions of axial compression, shear, torsion and bending, with both static and dynamic loading (Markolf, 1970; Wilder, Pope, and Frymoyer, 1988; Kazarian, 1975); impedance (Kazarian, 1972)); visco-elastic behaviour (Kazarian, 1975); damping (Markolf, 1970) and dynamic stiffness (Crocker and Higgins, 1967; Kazarian and Graves, 1977).

In vitro studies provide only limited information on the behaviour of the spinal column in the intact human. For example, the stiffness, damping and resonance frequency of the intact spine will be influenced by torso mass and muscle tension, and cannot be determined from in vitro studies. Although basic data on the ultimate strength and fatigue failure characteristics of the spine can be obtained from in vitro measures, knowledge of spinal loading and motion in vivo is required to relate material properties to the demands of normal activity, and extreme environments such as load carriage, vibration and impact.

Investigators have adopted two distinct approaches to study the biomechanics of the spine in vivo: direct measurement; and biomechanical analysis based on external loading, tissue properties and anatomical structure.

Direct measures of spinal loading have been achieved by measurement of intra-discal pressure in vivo (Nachemson and Morris, 1964), while motion of the vertebra in response to whole-body vibration has been studied by insertion of steel pins to the spinous process (Pope et al. 1991). Visco-elastic compression during spinal loading has been studied non-invasively by precise measures of stature (Corlett et al. 1987). Invasive measures of spinal loading and motion (Nachemson and Morris, 1964; Pope et al. 1991) are complex and have been limited to a few subjects. Nevertheless, these investigations provide an important data source for validating biomechanical analyses of spinal loading and motion, and for the prediction of acute and chronic failure. Thus, to obtain an understanding of the behaviour of the spine under conditions of dynamic loading in vivo requires the integration of information acquired through all three avenues of discovery: mechanical properties measured in vitro, loading and motion measured in vivo, and biomechanical analysis.

A detailed investigation of the mechanical properties of the spine in vitro was reported by Markolf (1970). In vitro specimens taken from the thoracic and lumbar spine were subject to static force-displacement testing in axial compression, shear, torsion, flexion and lateral bending. Results showed that the force-displacement curves were non- linear in compression, torsion and bending, but linear in shear. The response of thoracic spinal units to axial compression was more linear than that of the lumbar units, which displayed marked non-linearity at lower loads. However, there was no significant difference in the stiffness of thoracic and lumbar units measured at higher loads (Markolf, 1970). Compression characteristics provided good agreement with the results of Brown, Hansen and Yorra (1957), and Virgin (1951). Stiffness in axial compression was measured to be 1200 to 3300 $N.mm^{-1}$ at moderate loads of 200 to 600 N. Stiffness in axial tension was half of this value, while stiffness in shear was 10 to 15% of that in axial compression (100 - 500 $N.mm^{-1}$). The initial rotary stiffnesses for lateral bending, flexion and extension were all of similar magnitudes (0.7 to 4.7 Nm/degree) and did not vary significantly between lumbar and thoracic regions. Stiffness in axial torsion showed a discontinuity between the tenth and twelfth thoracic vertebrae, with a much greater stiffness occurring in the lower thoracic and lumbar regions. Stiffnesses in axial torsion and extension were found to be dependent on the posterior facets and ligaments.

Crocker and Higgins (1967) measured the dynamic stiffness of L1 - L2 spinal units. The vertebrae were compressed at 0.1 and 0.7 $mm.s^{-1}$ to reversible levels, and finally at 4 $mm.s^{-1}$ to failure. Stiffness was found to increase at higher strain rates. The force-displacement curve was non-linear at a compression rate

of 0.1 $mm.s^{-1}$ and low loads, but became linear at loads above 1000 N. At compression rates of 0.7 and 4.0 $mm.s^{-1}$ the spinal units displayed linear characteristics with a stiffness of 6000 and 12000 $N.mm^{-1}$ respectively. At a low compression rate and moderate loads (< 1000 N) stiffness was comparable with the measures of Markolf (1970).

Kazarian and Graves (1977) investigated the mechanical properties of the isolated vertebral centrum subject to axial compression. Results showed that ultimate load (yield point) and stiffness varied with position in the spinal column, and strain rate. At low strain rates (0.09 $mm.s^{-1}$) the ultimate load increased from 2700 N at T1 - T3 to 5600 N at T10 - T12. Both ultimate load and stiffness increased linearly with strain rate. In the T10 - T12 region ultimate load increased to 6900 and 8900 N at strain rates of 9 $mm.s^{-1}$ and 900 $mm.s^{-1}$ respectively. The corresponding stiffness increased from approximately 4700 $N.mm^{-1}$ at 0.09 $mm.s^{-1}$ to 7200 $N.mm^{-1}$ at the highest strain rate.

Henzel, Mohr and von Gierke (1968) provided a comprehensive review of vertebral compression due to axial loading based on in vitro observations. The authors identify four distinct events in the load deformation data of the spinal unit:

- end plate fractures
- proportional limit
- yield point
- total failure

End plate fractures have been observed to occur within the linear portion of the load deformation curve (Perey, 1957). The proportional limit defines the limit of linear elastic behaviour, beyond which load-deformation becomes non-linear and there is a reduced stiffness. It represents the point at which a material begins to fail, but is able to recover its preload form on release. The yield point defines the ultimate or maximum load beyond which irreversible deformation occurs. Total failure defines the point at which the structural integrity is lost and the material collapses.

The proportional limit and yield point of spinal units (vertebra-disc complex) and isolated vertebra in axial compression have been measured by Ruff (1950), Perey (1957), Yamada (1970), and Kazarian and Graves (1977). Ruff (1950) reported yield points of 5800 N to 10500 N for thoracic and lumbar vertebrae, (T8 to L5), with a progressive increase in strength with descending position in the vertebral column. These values are approximately 50% greater than those reported by Yamada (1970) and Kazarian and Graves (1977), but lower than those of Gozulov et al. (1966).

The mechanism of compressive deformation and failure of the vertebra-disc complex has been reported by several investigators (Brown, Hansen, and Yorra, 1957; Roaf, 1960; Perey, 1957; Brinckmann, 1985; Brinckmann, 1988). Axial compression of the spinal unit results in a loss of height measured between the vertebrae. Although there is evidence of decreased disc volume in the initial phase of compression (Brown, Hansen, and Yorra, 1957; Roaf, 1960), as the disc material itself is essentially incompressible, further height decrease must result in a radial bulge of the disc. Measurements by Brinckmann (1988) indicate an increase of disc compressive load from 1000 to 2000 N caused a disc radial expansion of 0.2 mm. Radial disc bulge is accompanied by a corresponding axial disc bulge, or more correctly, inward deformation of the vertebral end plates (Henzel, Mohr, and von Gierke, 1968; Brinckmann, 1988). Axial bulge has been reported to be of the order of 0.2 to 0.5 mm under physiologically safe loads, increasing to 1.0 mm at yield point (Brinckmann et al. 1983; Rolander and Blair, 1975). Thus, as the rims of the vertebra approach, the end-plates deform in the opposite directions such that the height at the centre of the disc remains almost unchanged (Brinckmann, 1988).

The relation between radial and axial disc bulge will depend on the deformability of the disc, and the stiffness of the end plate and underlying trabercular bone. At yield point, a deformation of 1.0 mm of the end plate will produce a mean compressive strain of approximately 6.6% in trabecular bone, which approximates the yield strain for that material (Brinckmann, 1988). Brinckmann (1988) also notes that axial overload, and repetitive loads below the yield point damage the end plates and adjacent trabecular structure, whereas the annulus remains intact unless the physiological limits of flexion or bending are exceeded. This suggests that disc herniation is caused by fatigue failure of the disc structure, (due to repetitive loading within the elastic limit of the material) rather than by a single mechanical overload (Brinckmann, 1985; Brinckmann, 1988).

Nutrition and Fatigue Failure of the Spine

Although compression fracture is the common failure mode of the vertebra-disc complex in severe axial impact loading, this mechanism does not apply to repetitive loading considered to be within the linear portion of the stress-strain curve. Low back pain and back disorders which have been associated with exposure to whole-body vibration and repeated shocks point to a chronic degeneration of tissues rather than acute failure (Sandover, 1981).

Two mechanisms have been proposed to relate vibration exposure to degenerative changes of the spine: impairment of nutrition; and mechanical fatigue due to repetitive loading (Dupuis and

Zerlett, 1987; Hansson and Holm, 1991; Brinckmann, 1985; Sandover, 1988).

Hansson and Holm (1991) provide a detailed discussion of the mechanisms of disc nutrition and speculate that nutrition may be disrupted by vibration. The authors identify two mechanisms for transfer of nutrients, molecular diffusion through the tissue matrix, or fluid transfer due to the pumping action of loading and unloading the disc. These mechanisms act across both the annulus fibrosis and the cartilaginous end plates of the disc. The authors state that the cause of disc degeneration is multifactorial, but that in addition to ageing, mechanical factors may initiate degenerative processes in the lumbar discs. A possible mechanism for this process is the disruption of blood flow in vessels surrounding the annulus fibrosis and under the end plate cartilage which will affect solute transport by both diffusion and fluid transfer to the disc (Hansson and Holm, 1991).

Kraemer, Kolditz and Gowin (1985) demonstrated that the disc acts as an osmotic system, releasing fluid under load until an equilibrium point is reached, and reabsorbing fluid as the load is released. This pumping action is said to play an important role in nutrition of the disc. Hence they conclude that continual alteration between loading and unloading which occurs in normal activity promotes the disc metabolism, whereas constant pressures, such as when sitting, lead to an interruption of the transfer of liquid and nutrients. Holm and Nachemson (1983) studied the effects of canine spinal movement during exercise on transport and metabolic parameters of the disc. They concluded that movement gives rise to positive nutritional variations. In a later study, these authors observed a reduced nutrient supply and loss of disc height in the spines of pigs exposed to vibration (Holm and Nachemson, 1983).

The role of mechanical fatigue as a factor in chronic degeneration of the spine has been proposed by Sandover (1983), Dupuis and Zerlett (1987), Brinckmann (1985) and Troup (1975). Troop (1975) points to evidence of fatigue failure of the neural arch in athletes subjected to repetitive loading. Brinckmann (1985) proposes that disc herniation is caused by fatigue failure of the disc structure rather than by a single mechanical overload. In support of this argument Brinckmann (1985) observes that clinical symptoms of disc herniation are caused by detached pieces of annular material and sometimes fragments of cartilaginous end plate.

Sandover (1983) proposed two hypotheses to relate fatigue induced failure of vertebral tissue to disc degeneration. In the first, dynamic compressive loading of the joint leads to fatigue induced microfractures of the end plate or subchondral trabeculae. Callous formed during the repair process leads to reduced nutrient diffusion. This hypothesis is supported by Brinckmann (1985), who states that although fresh end plate

fractures are not readily seen on radiograms, ossifications within the vertebral body as a late indicator of these events show that end plate fractures are rather frequent. In the second hypothesis, dynamic shear, bending or rotational loading of the joint leads to fatigue induced failure within the annulus, either as tensile failure of the collagen fibres, or as failure of cohesion between fibres or lamellae. Sandover also suggests that this process may relate to the annular lamellae already weakened by impaired nutrition as described in his first hypothesis.

The fatigue characteristics of bone have been studied by Lafferty (1978), and Carter et al. (1981). Lafferty (1978) derived relationships between fatigue life of bone (number of cycles to failure), stress frequency and peak stress. Results indicated that at frequencies below 30 Hz, fatigue is independent of stress frequency. At higher frequencies (for a constant level of peak stress) fatigue life increases with frequency according to the relationship:

$$N = A_1 \cdot f^a$$

where: N = number of cycles to failure

f = stress frequency

A_1, a = constants

At frequencies below 30 Hz the fatigue life varied only with peak stress according to the relationship:

$$N = A_2 \cdot S_p^b$$

where: S_p = peak stress

A_2, b = constants

Based on results of various studies employing bovine and human bone, Lafferty (1978) developed a predictive equation of fatigue life based on ultimate strength (S_u), applied (peak) stress (S_p), and stress frequency (f).

Carter et al. (1981) studied the fatigue characteristics of human cortical bone in axial loading. Their results suggested a fatigue strength of cortical bone of 7 MPa at 10^7 cycles, which is lower than that reported by Lafferty (1978) (40 MPa at 10^7 cycles). Carter et al. (1981) point out that previous fatigue studies were based on rotation and bending. They suggest that uniaxial fatigue behavior is more important than bending fatigue behavior in relating in vitro results to in vivo conditions.

Sandover (1983; 1985) proposed a model of fatigue-induced failure of the intervertebral joint in response to cyclic loading. Sandover utilizes the data of Lafferty (1978) and

others (Carter et al. 1981; Weightman, 1976) to develop the following relationship for fatigue:

$$N = (S_u/S_p)^x$$

where N = number of cycles to failure, S_u = static failure stress, S_p = applied repetitive stress, and , and x = constant. Sandover (1983) noted that the value of exponent x varied between biological tissues and test methodologies from x = 5 for cortical bone (Carter et al. 1981)) to 20 for cartilage (Weightman, 1976). Sandover (1983) proposed an exponent value of 9.95 based on Lafferty (1978). This value was revised to 7.7 in a later paper (Sandover, 1985). Sandover (1983; 1985) estimated the spinal loading in response to whole-body vibration at 2.0 $m.s^{-2}$ rms and 5 Hz, to predict the fatigue failure life of the vertebral joint. Fatigue failure was predicted to occur at 1000 working days for an exponent of 8 and following a single day of exposure for an exponent of 5.

Sandover (1985) extends his concept of fatigue induced failure by application of the Palmgrem-Miner hypothesis to obtain a dose-response relationship. This hypothesis, (Miner, 1945), states that the degree of fatigue damage is given by the summation of n_i/N_i, where n_i is the number of cycles at a particular stress level, S_i, and N_i is the number of cycles to failure at that stress. The effect of a particular vibration environment can then be estimated in terms of a "dose" value as:

$$D = n_i(S_i/S_u)^x$$

where D = fatigue dosage index

In this system, a dosage value of D = 1.0 represents the accumulated exposure at which fatigue failure is expected.

Loss of Stature: Spinal Creep

It has been reported that people lose approximately 1% of stature during the course of a day (Reilly, Tyrrell, and Troup, 1984). This loss of stature is then regained during the hours of sleep. Loss of stature occurs primarily through change in height of the intervertebral discs, and is exponential in form, the most rapid change occurring during the first hour after rising (Corlett et al. 1987). Various investigators have shown that the decay in stature can be accelerated by such factors as static spinal loading, dynamic lifting, running, and different types of seating. Lifting a 50 kg load repeatedly for 20 minutes would cause a shrinkage equivalent to the entire diurnal loss of stature (Tyrrell, Reilly, and Troup, 1985; Corlett et al. 1987). By comparison, sitting on a stool caused a height loss of 3 $mm.hr^{-1}$, and sitting in an office or easy chair with back support caused no height loss over 90 minutes (Eklund and Corlett, 1984).

It is probable that this difference is due to variations in posture and muscle activity, which will produce changes in spinal load. Evidence of a strong relationship between spinal load and height loss has led researchers to investigate whether stature loss can be used as an index of the effects of whole-body vibration. Results are inconclusive with some researchers reporting a height loss in response to vibration, and others reporting a height gain (Haslegrave, Shearing, and Corlett, 1989; Bonney and Corlett, 1988; Klingenstierna and Pope, 1987).

Haslegrave, Shearing and Corlett (1989) measured a height gain of 0.8 to 6.1 mm in response to one hour of vibration at 4 Hz and 1 $m.s^{-2}$ magnitude. Bonney and Corlett (1988) also report a gain of 1.9 mm in a one hour exposure at 4 Hz and 1 $m.s^{-2}$, but a loss of 1.0 mm with no vibration, and no change at 6 and 8 Hz. Sandover et al. (1991) have reported a gain in height of 1.1 and 1.9 mm following 30 minutes of sitting with and without vibration. The subjects returned to their pre-exposure height within 10 minutes of the end of exposure. Results of Sandover et al. (1991) do not support the concept of an inflammatory response proposed by Sullivan and McGill (1990). In contrast to the above, Klingsterna and Pope (1987) and Sullivan and McGill (1990) have reported a loss of stature in response to vibration exposure.

The conflicting nature of results may arise from the difficulty in obtaining accurate measures. The results are sensitive to the exact conditions of posture and timing under which measures are obtained immediately prior to and after vibration exposure. For example, when measures are taken in an erect posture after a seated exposure the effect of heel pad compression, reported to average 4.5 mm over 90 seconds, may confound results (Haslegrave, Shearing, and Corlett, 1989). Similarly, the large loss of stature reported by Sullivan and McGill (1990) who measured sitting height in response to vibration, may derive from visco-elastic compression of soft tissues of the buttocks, rather than the intervertebral discs. Hence, until more consistent data become available, and an explanation of stature change in response to vibration is elucidated, it is unlikely that the measure of stature will prove a useful means of estimating the effects of vibration and impact on the spine.

Impact Acceleration and Spinal Injury

Investigations of impact accelerations on the human body include horizontal seated impact occurring in vehicle collision, (Aldman, 1962; Glaister, 1978), vertical impacts occurring in the seated posture due to pilot ejection (Jones, Madden, and Luedeman, 1964), or lifeboat free-falls (Nelson, Hirsch, and Magill, 1988) and in the standing posture due to blast in ships (Hirsch and White, 1964). This review is limited to vertical

seated impact. Studies of impact acceleration include both cadaver and epidemiological studies.

Jones, Madden and Luedeman (1964) reported vertebral fracture incident rates of 21% occurring in 165 pilot ejections from United States Naval (USN) aircraft. The ejection seat was specified to produce accelerations of 18 to 22 g (180 to 220 $m.s^{-2}$) with onset rates of 180 to 380 $g.s^{-1}$. However, the authors point out that injury data and subsequent testing suggest a much wider range of acceleration. Comparable data obtained from British and Swedish aircraft using the same system showed a fracture rate of 19% in 220 ejections (Fryer, 1961) and 43% in 7 ejections (Hirsch and Nachemson, 1961). A 25% incidence rate was also reported in 55 cases of ejection using a SAAB seat (Hirsch and Nachemson, 1961). Laurell and Nachemson (1963) reported no vertebral fractures in 23 cases of ejection with accelerations of 15 to 20 g, and a 41% fracture rate from ejections at 20 to 25 g. Although fractures were found at all levels of the thoracic and lumbar spine, the USN and British data showed the highest rates occurred in the lower thoracic region from T8 to L1 (Jones, Madden, and Luedeman, 1964; Fryer, 1961). This region has also been found to be the most common injury site in cadaver impact studies (Myklebust et al. 1983). It is notable that over 50% of pilots injured by ejection sustained more than one fracture, suggesting a large variance in either impact acceleration or subject sensitivity.

Tolerance of the spine to G_z impact acceleration has been estimated by Stech (1963) from a combination of in vitro data of yield strengths of individual vertebrae, biomechanical analyses of the spine, and probability theory. Stech (1963) reanalysed the data of Ruff (1950) and Perey (1957) to construct injury probability curves as a function of z-axis acceleration level. Data included probability of end plate fracture, proportional limit deformation, and compression fracture at different vertebral levels. Stech and Payne (1963) also calculated the combined injury probability function of compressive fracture of a vertebra for the complete spinal column. Results indicated a 0.5 probability of fracture at an acceleration of 18 g at age 20, reducing to 13 g at age 40. By comparison the 0.5 probability of end plate fracture in the lumbar region was calculated to occur at approximately 10 to 12 g which represented about half the acceleration level for vertebral fracture.

There are several weaknesses in the analytical approach of Stech (1963) to the assessment of injury risk. The analysis does not include the effects of dynamic response, acceleration profile, pulse duration, spinal flexion, or loading of the articular facets, which will influence the probability of spinal injury (Henzel, Mohr, and von Gierke, 1968; Prasad, King, and Ewing, 1974; Glaister, 1978; Payne, 1991). Thus, although the predictions of Stech (1963) for individual vertebrae have been shown to provide reasonable agreement with epidemiological data (Henzel, Mohr, and von Gierke, 1968; Jones, Madden, and Luedeman,

1964), in general the epidemiological data indicates a higher tolerance level than the injury probability functions for the complete spinal column (Stech, 1963; Stech and Payne, 1963).

The role of the articular facets in impact acceleration was studied by Prasad, King and Ewing (1974) using instrumented human cadavers. The authors show that in response to axial impact, the facet joints are initially subjected to compression followed by a tensile phase. The latter results from a forward flexion of the head and torso, which applies a tension to the posterior structures of the spinal column. The forward flexion of the torso also causes increased loading of the anterior aspects of the vertebra, and anterior wedge fractures. Ewing, King and Prasad (1972) demonstrated that when hyper-extension of the spine was induced in cadavers the acceleration level of fractures was raised from 10 g to 18 g. Prasad, King and Ewing (1974) conclude that anterior wedge fractures result from eccentric compression coupled with unloading of the facets. An increase in tension of the posterior structures will cause the vertebral body to sustain a greater load (due to a bending moment) than the applied axial force. In the hyper-extended mode, the facets relieve the vertebral bodies of compressive stress by creating a dual pathway for load transmission.

Hodgson, Lissner and Patrick (1963) investigated the effect of rate of onset of acceleration (jerk) on spinal response to impact. Human cadavres were subjected to acceleration and jerk up to 18 g (180 $m.s^{-2}$) and 2600 $g.s^{-1}$ (26000 $m.s^{-3}$) respectively. Strain gauges and accelerometers were attached to the vertebrae. It was found that the dynamic load factor (peak/mean acceleration response) and peak strain rate increased with the rate of onset of acceleration, up to 1000 $g.s^{-1}$. Above this acceleration rate, the dynamic load factor remained independent of rate of onset of acceleration.

Glaister (1978) points out that because of the dynamic characteristics of the human body, the peak force transmitted to the body tissues is dependent on impact acceleration magnitude, pulse duration, and natural frequency of the body (f_n). For a constant acceleration, or for accelerations with slow onset rates and durations greater than f_n, the initial response is equal and opposite to the applied force with a transfer factor of 1.0 (i.e. equilibrium is established). If the pulse length is much shorter than the natural period of the body, the elastic tissues will still be compressing when the acceleration ends. Under these circumstances, (in a simple mass-spring system), the force transmitted is then dependent on the imposed velocity change (Glaister, 1978). As velocity changes of an acceleration pulse can be represented as the integral of acceleration with respect to time, then for equal levels of stress (spring compression) the pulse amplitude (acceleration) will increase linearly with the inverse of pulse length.

In situations where the acceleration pulse approximates the natural frequency of the body, the dynamic response of the body causes a lag, followed by an overshoot in acceleration response (i.e. a transfer factor > 1.0). Glaister suggests that for a seated posture, where the natural frequency of the body approximates 5 Hz, the critical pulse length for impacts is about 0.2 seconds. This value corresponds closely with the pulse length of early catapult ejection seats (Jones, Madden, and Luedeman, 1964). Glaister extends his theoretical approach to produce curves of equal acceleration tolerance as a function of pulse duration, for the body in seated, standing and supine postures. The author emphasises that these curves should be treated with caution as they are only intended as an indication of the most probable level of tolerance.

Von Gierke (1972) discusses the components of the body which contribute to the main or identifiable resonance frequencies and proposes limits for impact tolerance based on acceleration magnitude and pulse duration. For very short pulses (< 10 ms) the head is considered to be the limiting system; between 10 and 100 ms the lumbosacral system is most sensitive, and above 100 ms the spine-abdomen becomes the critical region. The composite tolerance curve of von Gierke is based on the resonance frequencies of approximately 5, 10 and 30 Hz for the spine-abdomen, lumbosacral system and head respectively and otherwise follow the same acceleration-pulse duration profile as adopted by Glaister (1978).

Although some authors have concluded that the human response can be approximated by a linear system (Sandover, 1982; Cole, 1978), Payne (1991) suggests that the dominant natural frequency of the body increases from 5 Hz when exposed to whole-body vibrations to approximately 12 Hz when subject to severe impact. The shift of frequency is attributed to a pronounced non-linearity of human structure which results in increased stiffness and natural frequency at greater acceleration magnitude (Payne, 1991; Weis and Mohr, 1967).

Conclusions

The spine is a complex structure consisting of a series of rigid elements (vertebrae) connected by flexible visco-elastic units (intervertebral discs). Compressive, bending and shear loading can be transmitted by a combination of forces in the intervertebral discs, apophyseal facet joints, ligamentous structures and active muscle contraction. The intervertebral disc is subject to stress whether a person is standing, walking, or seated. An important difference between a purely mechanical system and a living system is that the mechanical system does not change under constant stress provided the strain does not exceed the elastic limit. In a biological system the elastic properties of tissue are a time dependent function of the applied stress.

Thus, loss of fluid takes place from the intervertebral disc space in response to static loading, (referred to as creep). This will affect the stability of the spinal unit and cause a redistribution of stresses in the surrounding tissue. In addition, unlike mechanical structures, the properties of biological materials are also a time dependent function of their nutritional status.

Investigations of spinal units in-vitro have shown non-linear load-deflection characteristics. Ultimate strength and stiffness increases with the rate of compression. Fracture of the end plate occurs within the elastic limit of the material. In general, failure occurs due to compressive fractures of the vertebra, while the intervertebral disc remains intact. In single impact studies, vertebral damage occurs most frequently in the lower thoracic and upper lumbar region in the form of anterior wedge fractures, at impact accelerations of 18 to 25 g. Both nutritional and fatigue mechanisms have been postulated to explain chronic degenerative failure in response to WBV.

Calculation of fatigue failure properties are based on in-vitro data. They do not include consideration of the ability of tissue to recover or repair through on-going nutritional mechanisms. Hence, these calculations may underestimate the real fatigue life of tissues in-vivo due to the absence of a regenerative model.

Although there appears to be distinct differences in the mechanisms of acute and chronic injury, it is probable that both types of injury are a function of material behaviour. Thus, by studying the material properties of tissue, it may be possible to devise a unified theory for mechanical injury which will encompass both the mechanisms of acute impact injury and chronic degenerative failure.

Biodynamic Models

Introduction

During the past 40 years, numerous attempts have been made to model the biodynamic characteristics of the human body. These models have varied from relatively simple mass spring models containing one degree of freedom (Payne, 1991) to highly complex representations of the human body containing multiple degrees of freedom (Hopkins, 1972) and capable of simulating 3-dimensional motion (Amirouche and Ider, 1988). The development of increasingly complex models has paralleled the development of the computer, and the increasing availability of detailed experimental data on which to base model parameters.

A fundamental objective of biodynamic models is to predict the behaviour of the human body (or individual tissues) in response to vibration or impact. The output of the model (i.e. behaviour) may be expressed in terms of displacement, acceleration, stress or physiological response. Thus, a well developed biodynamic model could prove to be an ideal tool for assessing the health effects of impacts and vibration. To achieve this goal it is first necessary to establish the requirements of such a model. A feature of most models, regardless of complexity, is that they have been shown to be valid when compared to experimental data on human response. This poses the question of whether there is a need for more complex models.

Biodynamic models can be classified according to either the purpose of the model, or the function to be modelled (von Gierke, 1971). The most common purposes of models are:

- To understand basic pathological processes, physiological responses, or biomechanical responses to various mechanical stresses.
- To predict human response to stress in circumstances where experimental data is either unavailable or unobtainable.
- To determine the engineering design of systems to provide protection, comfort or safeguard performance of the operator.

Models have been classified according to function as: kinematic models; total body response models; subsystem models; and tissue property models (von Gierke, 1971). Within the above classifications, models may be designed to assess the effects of axial acceleration, horizontal impact (e.g. vehicle collision), vibration (uniaxial or triaxial), and blast. The fact that most models are designed to fulfil a specific purpose or examine a

specific function (within a specific environment) goes some way towards explaining the proliferation of models reported in the literature.

Von Gierke (1971) reviews the development of early biodynamic models. Various aspects of human modelling are discussed including lumped parameter dynamic response models and their application to acceleration, impact and blast. The author notes that attempts to derive model specific injury modes, has led to more detailed and specialized models such as lumped parameter, discrete parameter, and continuum models of the spine, or models considering non-linear behaviour of tissue. Of these more complex models, which include axial, shear, and bending deformations, von Gierke (1971) finds that the state of modelling is ahead of the relevent experimental data. He concludes that to improve the operational usefulness of the simple lumped parameter model such as the dynamic response index (DRI) (Air Force, 1970), the incorporation of x and y-axis acceleration appears to be more important than further refinement of z-axis criteria.

The inability of uniaxial lumped parameter models to provide insights into spinal injury mechanism was highlighted by the work of Orne (1969) and Orne and Lui (1971). Orne and Lui (1971) investigated the effect of impact accelerations on the spinal column in three independent motions; z-axis translation, x-axis translation, and y-axis rotation. Their models simultaneously account for axial compression, antero-posterior shear, saggital plane bending deformation of the intervertebral discs, the viscoelastic behaviour of the discs, the variable size and mass of the vertebrae and discs, spinal curvature and eccentric inertial loading. A discrete parameter model is used with individual visco elastic elements for the intervertebral discs. A 10 g (100 $m.s^{-2}$) impulse was applied to the model with acceleration rise times of 5 and 14 ms. Analyses were repeated with the model restricted to uniaxial motion. Results showed that the uniaxial (compression) model provided substantially different results from the model in which shear and bending stresses were included. Large bending moments occurred in the thoracic region which together with axial force produced a high compressive stress in the anterior aspect of the thoracic vertebrae. The authors noted that injury data from pilot ejections also indicated a high incidence of fractures in the thoracic vertebrae (Hirsch and Nachemson, 1961). The addition of a horizontal (x-axis) component to the acceleration (pulse vector 10^{o} from z-axis) was found to produce a whipping action, which substantially increased shear and bending stresses in the lumbar region, and decreased axial response along the vertebral column (Orne, 1969). Orne (1969) suggests that this latter effect may explain the difference in injury reported for impulse ejection (often thoracic damage) compared with whole-body vibration of vehicle operators (primarily lumbar injury).

A three-dimensional discrete parameter model was reported by Panjabi (1973). Unfortunately this paper was restricted to a

general theory with no parameter values provided and no validation of the theory.

Further insight into the kinetics and kinematics of spinal response to impact was provided by Prasad and King (1974). The authors surveyed existing spinal models and commented on inadequate validations and the inability of most models to describe conditions of load transfer from one vertebra to the next. They legitimately question the underlying principles and validity of simple (lumped parameter) axial models that employ a transfer function to estimate spinal compressive load. A discrete parameter model of the spine is presented in which each vertebra has 3 degrees of freedom in the saggital plane (z and x-axis translation and y-axis rotation). Thus, configuration of the model permits calculation of axial compression, shear, and bending stresses. A new feature introduced in the model, which differentiates it from those of Orne and Liu (1971) and Braunbeck and Wilkinson (1981) is the transmission of load through the articular facets, based on experimental work using cadavers (Prasad, King, and Ewing, 1974). Results of the model provided a good correlation with experimental results for different impact accelerations and spinal postures. The model showed very little evidence of dynamic overshoot of the form predicted by axial lumped parameter models. Peak forces occurred late in the acceleration pulse and were a result of head and torso kinematics and load transfer. In common with Orne and Liu (1971), Prasad and King (1974) found that the anterior aspect of the vertebrae were subject to extreme loading during the second phase of the impulse, when the facets unload and go into tension.

A sophisticated discrete parameter model of the spine was also developed by Belytschko and Previtzer (1978). Their model included the head, vertebrae and ribs interconnected by deformable elements representing the discs, ligaments and viscera. Four models of differing sophistication were described ranging from a complex spine model (CSM) with 252 degrees of freedom to a simplified spine model (SSM) having 32 degrees of freedom. Outputs from the models gave good agreement when compared with human impedance data. The authors concluded that the primary resonance frequency of impedance curves obtained from human subjects results from a combination of buttock-seat resonance, flexural response of the spine and visceral resonance. The model was further developed to include an injury criterion based on stresses in the vertebral bodies resulting from axial compression and bending. Statistical data on the compression strength of bone derived from experimental sources is used to construct a probability function to predict injury risk at discrete levels of the spine. This model and the corresponding injury criterion is primarily concerned with destructive impact and compressive fracture of the vertebrae. Hence, it does not address the risk of fatigue-induced injury due to repetitive impact experienced in vehicle operation.

Muksian and Nash (1974) reported a uni-axial lumped parameter model of a seated human with individual elements representing the head, vertebral column, upper torso, thorax, diaphragm, abdomen and pelvis. The authors extended the complexity of the lumped parameter model by inclusion of non-linear stiffness and damping properties for the torso, thorax and abdomen complex. Results demonstrated that a better agreement was obtained with experimental data by inclusion of the non-linear elements. While non-linear tissue characteristics proposed by Muksian and Nash (1974) are an improvement in the sophistication of theoretical models, other authors have demonstrated equally good results with linear models (Payne, 1991). The model (Muksian and Nash, 1974) exhibited severe discontinuities in the spinal and torso acceleration transfer functions which remain unexplained. In a subsequent publication, Muksian and Nash (1974) report a revised 3-dimensional lumped parameter model to provide further evidence of non-linear (frequency dependent) damping properties in human response to vibration. Parameters of the model were adjusted until acceleration ratios converged to existing experimental data. The resultant model exhibited linear characteristics up to input frequencies of approximately 6 Hz. Between 10 Hz and 30 Hz, visceral damping was found to vary parabolically with frequency. The frequency-dependent characteristics are justified since during vibration, the primary resistance is muscular reaction, which is known to be frequency dependent. To obtain a satisfactory fit to experimental data the authors were obliged to assume linear properties for frequencies less than 10 Hz and introduce non-linear properties for frequencies greater than 10 Hz. The model represents a mathematical fit of experimental data rather than a physiological model including active muscle properties.

Most spinal models, regardless of sophistication, treat the musculature as passive viscoelastic elements. A more physiological approach is reported by Bluthner, Hinz and Seidel (1986) and Seidell, Bluethner and Hinz (1986). The authors describe a biomechanical model in which the compressive load at the L3/L4 disc is calculated from measures of trunk acceleration, upper torso mass and EMG activity. The transmissibility between T5 and the seat are measured at peak (maximum and minimum) accelerations. An output of muscle force, ligament force, and disc compressive force with time is provided by the model. At 3 $m.s^{-2}$, and frequencies of 1 to 7 Hz, disc compressive forces ranged from 2 to 4.5 kN. The main conclusions of this study are that muscle activity does not protect the intervertebral disc from stress at most frequencies, and that the magnitude of compressive forces do not correlate uniformly with vibration intensity. The authors also calculated that the magnitude of stress at this vibration amplitude was sufficient to cause fatigue fractures of the cartilaginous end plates.

A spinal model that gained popularity and was the basis for a standard on seat ejection tolerance limits (Air Standardization Coordinating Committee, 1982), was the Dynamic Response Index

(DRI) developed by Payne (Nachemson and Morris, 1964). The DRI, is obtained by applying the acceleration-time pattern to a mass-spring-damper analogue of the spine (Nachemson and Morris, 1964). Allen and Payne further developed the model over the years to account for not only single impacts, but an increasing number of impacts in a 24 hour period (See section on Standards and Guidelines for more details). The DRI was validated with spinal injury from aircrafts. Payne, in an unpublished document (Payne, 1991), converted the DRI to rms units for comparison with the ISO Standards (See Appendix B, Table B-12). The severe discomfort boundary of the DRI for a single shock (4 to 9) corresponds to the 5 g plateau acceleration of Glaister (1978). For 10 shocks per day, the peak input according to the DRI conversion is 3 to 6.5 g. Although used for seat ejection, the DRI and subsequent curves developed by Allen and Payne were rarely used for other applications. In 1986, Anton looked at 223 ejections and calculated the DRI values. He found the DRI to be a poor predictor of injury (Sandover and Dupuis, 1987).

Coermann et al. (1960) measured abdominal wall displacement, thoracic expansion and oscillating air volume in supine humans subjected to axial (z-axis) vibration. The authors produced biodynamic models of the thorax-abdomen system. Models were presented in the form of electrical analogues with different configurations to represent externally applied forces due to whole-body vibration, transthoracic pressure (such as occurs in a Drinker respirator), and slow blast wave decompression. The use of an electrical analogue model facilitates measurement of tissue stresses in the form of node voltages within the circuit. It is surprising that few researchers have pursued this avenue of biodynamic modelling given the obvious potential for constructing complex discrete parameter tissue models with simple output measures.

One of the most interesting models reported is a hydrodynamic model of cardiovascular function developed by Knapp (1974). The model was constructed in analogue computer form. The output was compared with animal experimental data and a previous mechanical model. All three approaches demonstrated a sensitivity to the phase relationship and frequency relationship between the input vibration frequency and the heart frequency. The models show that vibration alone can generate aortic pressure, and flow due to the presence of non-return valves in the system. The resultant pressure and flow waveform may be amplified or attenuated, depending upon the phase of the two signals. Peak aortic pressures are greatest at 2 to 4 Hz. At these frequencies, peak pressures of up to 3-times normal were predicted by the model when standing and exposed to a vibration amplitude of 30 $m.s^{-2}$. Peak aortic flows of 2-times normal were recorded at 4 to 6 Hz. In the seated posture, values were predicted to be 50% lower. With asynchronous vibration, aortic flow tends to be attenuated (by up to 90%) and amplified alternately according to the phase relationship. The authors observe that while maximum and minimum peaks in blood pressure

and blood flow may serve as general indices of cardiac stress, they do not provide information on whether cardiac output is increased, unchanged, or attenuated. No evidence is provided regarding health hazards of vibration on the cardiovascular system. While elevated aortic pressures can be considered a measure of stress, it has also been suggested that vibration can provide a beneficial effect of increased cardiac output. Vibration has been promoted in patients with cardiac insufficiency as an external cardiac assist device. Changes in pressures and flows were found to decrease rapidly above 4 to 6 Hz, and approached control values by 15 Hz.

In summarizing the status of biodynamic models, Griffin (1981) states that:

- Most models are based on inadequate experimental data
- Current models are highly restrictive in their application
- Models devised for different purposes (for example spinal injury or performance) may have little in common. The extent of human variability and shortage of experimental data make it possible for a one degree of freedom model (such as the DRI) to compete successfully with much more sophisticated 3-dimensional or discrete parameter models.

From the biodynamic models reviewed a number of shortcomings of existing models are apparent. Firstly, although many models are available to predict the transfer function of one or more body sub-systems, these models do not extend to the prediction of chronic health effects, or tissue damage, as a result of WBV or repeated impacts.

Secondly, most models are mathematical rather than physiological in nature. They consist, therefore, of mechanical analogues of the human body which can be represented mathematically for computational purposes. The model parameters are tuned to match known experimental data (such as the seat to head transfer function, total body impedance or apparent mass). This undermines the validity of the model because it is no longer independent of the experimental data with which it is compared (Amirouche, 1987). In this respect these models represent only mathematical solutions of human body response. They provide no information regarding the underlying physiological or biomechanical effects on the body sub-systems or tissues. To achieve this, a physiological or biomechanical model is required in which the parameters are based on known tissue properties.

Thirdly, most models have been validated on the basis of a single output response such as seat to head transfer function (Muksian and Nash, 1974; Braunbeck and Wilkinson, 1981). It has been shown that such response functions can be adequately

represented by a simple one or two degree of freedom system. Thus, this type of testing is inadequate to properly validate a more complex model in which the various subsystems of the body are represented (Muksian and Nash, 1974). In this context the complexity of current modeling has outstripped the capacity for experimental validation (Soechting and Paslay, 1973; Panjabi, 1973; Rizzi, Whitman, and DeSilva, 1975).

Fourthly, many of the models, including some of the more complex mathematical models, are restricted to uniaxial displacement (Payne, 1965; Muksian and Nash, 1974; Demic, 1989). While this may be acceptable in representing impulse response, such as is experienced in pilot ejection, it places a serious constraint on any attempts to represent human response to whole-body vibration and repeated impact experienced by all-terrain vehicle operators. It has been demonstrated that in axial impact loading the principle area of damage is to the thoracic vertebra (Jones, Madden, and Luedeman, 1964), whereas epidemiological data indicate that in vehicle operators the most common area of damage is the lumbar vertebrae (Dupuis and Zerlett, 1987; Hansson and Holm, 1991). The latter effect may result from the introduction of an anterior-posterior component of acceleration, creating a flexor torque, and hence increased compression at the lumbar joints. These different effects have been demonstrated by Orne (1969) in a model simulating compression, bending and shear. Such differences in injury site cannot be predicted by a lumped parameter model, or a discreet parameter model restricted to uniaxial input.

Despite the criticisms, there have been several important contributions to biodynamic modeling which have direct relevance to the development of a health hazard index.

- Orne (1969) has shown that the introduction of anterior-posterior input forces, and the presence of bending moments (and hence flexion) within the spine, substantially alters the prediction of compressive stresses acting on the thoracic and lumbar motion segments

- Prasad and King (1974) have shown that the articular facets play an important role in the transfer of compressive forces during axial impact loading.

- Hinz and Seidal (1989) have shown that any fatigue model based on rms values of acceleration, as an estimate of input stress, will underestimate the health effects of vibration. This is due to the non-linear nature of the transfer function between the input acceleration at the seat and the output acceleration wave form.

- Sandover (1983) has proposed a model based on fatigue failure of materials. Sandover selected data on the fatigue characteristics of bone and cartilage to model fatigue failure

of tissue in response to cyclic loading. Models of both vertebral end-plate and the disc annulus suggested the possibility of fatigue failure in those structures.

- Seidel, Bluethner and Hinz (1986) constructed a model of stress in the lumbar spine based on anthropometric data, EMG activity and accelerations of the upper trunk (measured at the thoracic vertebra). The predictions of this model also supported the possibility of fatigue failure at the end plates of lumbar vertebrae after long term exposure to whole-body vibration.

Conclusions

In the development of guidelines for prediction of health effects of vibration, there are two possible approaches to be considered:

1. Subjective response model

2. Biodynamic response model

The current ISO Standards (2631,1982) are based on the former approach. This is possibly due to the limitations of early biodynamic models. However, subjective response models have also been widely criticised for their inability to accurately describe the chronic health effects of vibration and repeated impacts. In particular, there appears to be little evidence to support the time dependence of a subjective response model, and hence the time dependency of the current ISO Standard. Although a subjective response model may be acceptable for steady state vibration exposure, it becomes increasingly difficult to apply with confidence in the presence of multiaxial vibration or vibration with repeated impacts. In these circumstances it is probable that a biodynamic model can provide a more versatile prediction of human response. The Dynamic Response Index (DRI), designed to assess health effects of impact loading, is based on a simple biodynamic model. The advances achieved since the introduction of the DRI offer the potential of a more sophisticated biodynamic index incorporating the important features of more recent models, such as the consideration of compression, torque and shear forces, muscle activity, non-linear stiffness and damping, material properties and fatique characteristics.

Standards And Guidelines For Shock And Vibration

Introduction

Standards exist for a variety of purposes and applications. Ideally a standard governing environmental exposures, such as shock and vibration, should address three questions:

1. Who is at risk to which particular health conditions?

2. What combinations of exposure times and environmental factors will produce these conditions?

3. What are tolerable, accepted, or optimal environmental conditions in view of the effects? (Sandover, 1979).

Although a variety of guidelines exist, the main standards for human response to vibration have been developed by the International Organization for Standardization (ISO). Early versions of the ISO Standard (2631, 1974) defined vibration evaluation procedures and recommended tentative limits, although both the procedures and limits were somewhat ambiguous (Griffin, 1990). The emphasis in the recent Draft Revision of the ISO Standard and the British Standards (6841, 1987) is for unambiguous evaluation procedures and an absence of limits within the body of the standard (guidelines are given in an appendix). The philosophy is that the organizations responsible for enforcement should be setting the limits (Griffin, 1990). All standards require periodic updating; however, in the case of the ISO Standards, there has not been a major revision since 1982. Recent advances in the study of human response to impact and vibration, as well as improved computational techniques and procedures, are influencing the standards now under development.

This review will cover the main standards and guidelines governing vibration and shock. Each standard or guideline contains a summary of the experimental and computational techniques used. Where appropriate, a discussion of the limitations of the standard is included.

ISO 2631 (1974): Guide to the Evaluation of Human Exposure to Whole-Body Vibration

Work on this first international standard for whole-body vibration (WBV) began in 1966 among the 20 member countries (Griffin, 1990). The goals were to facilitate evaluation and comparison of data from continuing research in the field, and to give provisional guidance as to acceptable human exposure to whole-body vibration (International Standards Organisation, 1974). It was not intended to be a standard setting firm limits,

but rather a guide for evaluation of exposures with respect to human response (von Gierke, 1975). The ISO 2631 standard is the most universally applied WBV standard and was approved by all but two countries (U.S.S.R. and U.K.).

The vibration measurements are performed in three mutually orthogonal directions with linear accelerometers which are referenced to the standard biodynamic coordinate system. For a seated operator, vibration signals are measured at the interface to the human body with a triaxial accelerometer mounted in a special seat-pad. Careful choice of accelerometer type, signal conditioning and recording equipment is recommended to avoid problems related to frequency response, dynamic range and sensitivity. Calibration of all elements of the measurement chain is recommended but not described in the standard. The standard units used for translational vibration are ($m.s^{-2}$).

The minimum duration of vibration time history to be recorded is not stated in the standard. The record must be long enough to provide sufficient accuracy and resolution of the lower frequency components that may be contained in the signal. Clear documentation of all experimental conditions is required. The maximum duration of continuous exposure which are specified in the guidelines is 24 hours.

Computational Methods

The raw acceleration records are first processed using a one-third octave band frequency analysis (American National Standards Institution, 1986). The rms amplitude of the vibration signal within one-third octave bands is determined over the frequency range from 1 to 80 Hz, either by analog or digital methods. The resulting values are then multiplied by weightings for each frequency band to scale the signal to its relative effect on the human body. Weightings are provided for the x, y, and z axes translational vibrations.

No adjustment to the phase is made in the calculation. The overall rms and peak values are determined for the weighted (or filtered) signal. From these values the crest-factor is calculated, which is defined as the ratio of the peak value to the rms of the overall weighted signal. This standard is considered to be applicable for weighted records with crest-factors of up to 3.0.

The weighted 1/3 -octave rms values thus calculated are then plotted graphically. Limits for each octave band are specified which provide guidelines for comfort (Reduced Comfort Boundary, RC), fatigue-decreased proficiency (FDP) boundary, and health (Exposure Limit, EL). Where a limit, in any particular band, is exceeded, it signifies that the guideline is exceeded. For example, an exposure of 6 hours per day in the z-axis has a FDP limit of 0.36 $m.s^{-2}$, and an EL of 0.785 $m.s^{-2}$.

An alternative method of evaluation of broad band signals is suggested that involves determining the overall rms value of the weighted signal. In this case the weighted value is compared to the limit in the 4 to 8 Hz range for z-axis vibration and to the limit in the 1-2 Hz range for x and y axes vibration. Weighted rms values are denoted as a_{xw}, a_{yw} and a_{zw} respectively.

Limitations

It was not clear from the ISO Standards which data were used to develop the curves. Bobbert (1974) and Boileau (1988) discuss the lack of data for some guidelines; for example, the x and y limits were based on only two papers. Most of the studies were conducted in sinusoidal conditions, and the results may not apply to random broad-band vibration. The choice of the 1 to 80 Hz frequency range and shape of the frequency weighting has been questionned, as well as whether the same shape can be used to describe all three criteria of comfort, performance and health (Oborne, 1983; Boileau, 1988). It is assumed in the standard that any effects from rotational vibration can be accounted for by its translational counterpart.

The crest-factor was given an important status, although the definition was ambiguous (Boileau, 1988). Also, a crest-factor of 3 is found in almost all environments (Griffin, 1990). There was no method for evaluating signals with a number of repeated shocks imbedded in the acceleration time series. Furthermore, no guidance was provided for digital methods of data acquisition (e.g. sampling rates anti-aliasing filtering etc.).

When assessing complex or wide band spectra the overall weighted rms comparison and highest 1/3-octave band comparison may lead to quite different results. In addition, since filters are designed for the range of 1 to 80 Hz, it is not clear how to handle frequencies below 1 Hz and above 80 Hz.

Probably the most controversial aspect of the standard is the overly-complicated time-dependency. The curves suggest that effects from 1 to 4 minutes are independent of time, which is not the case with subjective data (Griffin, 1990). The curves were based on little or no evidence, and whether there are time-dependent effects, and their relationship with long-term health effects, is still questionnable (Clarke, 1979; Howarth, 1986).

ISO 2631-(1978); Amendment 1 (1982) and Addendum 2 (1982)

This revision attempts to eliminate the ambiguity between assessments and extend the range of applicability (International Standards Organisation, 1978).

Amendment 1 (1982)

Vibration with crest-factors up to 6.0 is included in the standard, and guidance is provided on the definition and measurement of this parameter. The primary measure of crest-factor involves frequency-weighted accelerations, but an initial screening can involve un-weighted accelerations. A minimum duration of one minute for the vibration signal is also specified.

Use of the overall frequency-weighted acceleration is recommended over the discrete 1/3 octave-band analysis for the assessment of comfort and fatigue-decreased proficiency. The time-dependency is simplified.

For vibration in more than one direction, the overall weighted vector sum (a_v) is to be calculated as:

$$a_v = [(1.4a_{xw})^2 + (1.4a_{yw})^2 + (a_{zw})^2]^{1/2}$$

Vector sums are compared with the frequency weighted acceleration limits for z-axis vibration in the 4-8 Hz range. The difference in sensitivity between x and y and z axes is accounted for by the coefficients in the above equation.

Addendum 2 (1982)

In this addendum, an extension of the standard is made to allow evaluation of vertical vibration in the frequency range 0.1 to 0.63 Hz. Vibration in this range is significant for motion sickness. Computation and analysis is similar to the one-third octave band frequency analysis with a separate frequency weighting for 0.1 to 0.63 Hz. An overall frequency-weighted acceleration is constructed.

Limitations

The phase characteristics of the frequency-weighting (and band-limiting) filters remain unspecified in this revision. Accordingly, there is no reason for measurements of crest-factor or overall accelerations to agree between investigators. For crest-factor determination, the minimum time of 1 minute may not be long enough, particularly when the signals contain repetitive jolts and impacts.

With respect to the motion sickness component of this standard, lack of data has prevented evaluation techniques from being presented for other axes which may be significant, such as pitch and roll. Also, no mention is made of the possible effects of impacts and jolts on the motion sickness limits.

ISO 2631/1 (1985) and 2631/3 (1985)

ISO 2631 (1978) was republished with an Amendment in 1985 as ISO 2631/1 "Evaluation of Human Exposure to Whole-Body Vibration - Part 1: General Requirements"(International Standards Organisation, 1985). Addendum 2 (1982) was issued as ISO 2631/3 "Evaluation of Human Exposure to Whole-Body Vibration - Part 3: Evaluation of Exposure to Whole-Body Z-Axis Vertical Vibration in the Frequency Range 0.1 to 0.63 Hz.". There were no substantial changes to the standards.

ISO 2631/2 (1989) Evaluation of Human Exposure to Whole-Body Vibration - Part 2: Continuous and Shock Induced Vibration in Buildings.

This standard describes methods and analysis techniques for evaluating the vibration environment in buildings (International Standards Organisation, 1989). It deals mainly with the perception of vibration in buildings. Measurement techniques and analyses are similar to those of ISO 2631-1, with identical weightings applied to the one-third octave frequency band results for x, y and z axes. Superimposed on the one-third octave plots, base curves are supplied which provide limits above which human annoyance is a factor.

Annex B of the standard lists state-of-the-art methods being investigated for more detailed characterization of impulsive vibration. The list includes peak methods, impulsive rms methods, root mean quad measures and response or shock spectra methods.

ISO/TC 108/SC4 (1991) Third Committee Draft Revision

The technical committee began a complete revision of ISO 2631 in 1979 to incorporate new research and epidemiological data, and introduce new methods. Changes include consideration of intermittent vibration and higher crest-factors, simplified time dependency, inclusion of vibration inputs from backrest, footrest and rotational axes, and removal of limits from the main body of the standard. This latest draft revision proposes significant changes to the computational methods involved. New techniques and weightings are similar to some of the analysis techniques of the British Standard 6841 (1987).

Computational Methods

The Third Committee Draft Revision applies to weighted vibrations from 0.5 to 80 Hz, with crest-factors up to 12, which incorporates most vibration environments. The rms measure

continues to be the preferred method of analysis for crest-factors up to 12 to maintain compatibility with existing databases. Six frequency weightings have been defined, which allow quantification of the relative effects of different vibration frequencies on health, comfort, perception and motion sickness. These different weightings are applied to the appropriate axes in a similar way as in previous ISO standards. A key development is the complete specification of all filters (magnitude and phase). This has an effect on the peak observed values of the weighted signal. Appropriate band-limiting filters are also provided. The time dependency shape has been altered as it was considered too severe for short durations and the 24-hour limit was considered too low. Due to lack of data, time dependency has been removed for comfort and perception. The fatigue-decreased proficiency boundary has been removed. The Draft states there is a documented effect of vibration on activity, but no relationship with increased time. The weighting for health (W_k) is similar to a_{zw} in the original ISO Standard, but evaluation of health should now also include vibration at the backrest and effects are assessed independently in each axis.

The weighted vector acceleration, a_v, is now defined as:

$$a_v = [(k_x a_{wx})^2 + (k_y a_{wy})^2 + (k_z a_{wz})^2]^{1/2}$$

where a_{wx}, a_{wy}, and a_{wz} are the frequency weighted rms accelerations in the x, y, and z axes respectively and kx, ky, and kz are defined multiplying factors.

Methods for evaluating rotational accelerations (rad./s^2) at the seat, translational accelerations at the footrest and acceleration at the seat back are described, together with appropriate weighting curves. Also, similar methods for evaluating the effect of vibrations on a recumbent person are included.

For the evaluation of health effects the new Committee Draft Revision specifies the normalized vibration dose value (NVDV), which is defined as,

$$NVDV = 1.4 a_w (T/Ts)^{1/4}$$

where a_w is the frequency weighted rms acceleration in the x, y, or z-axis, T is the duration in seconds, and Ts is a constant, 28,800 sec, representing the exposure time of 8 hours.

When the vibration exposure consists of two or more periods of exposure to different acceleration magnitudes, the normalized vibration dose value for the total exposure is calculated from the fourth root of the sum of the fourth powers of individual

dose values. The annex states there is insufficient data to show a quantitative relationship of the probability of health risk, but that disorders are probable at NVDV values over 1.3. Between 0.95 and 1.3 (Health Guidance Caution Zone), disorders can occur, and below this value there is insufficient evidence to indicate disorders (International Standards Organisation, 1991). The zone is similar to the former ISO exposure limit for 4-8 hours and the Draft suggests this level has been influenced by epidemiological data, while the level for shorter exposures (<30 minutes) has been influenced by subjective data. Data were interpolated between 30 minutes and 4 hours, and between 8 and 24 hours. When the crest-factor is above 12, the vibration dose value is the preferred measure. VDV is defined as:

$$VDV = (\int_0^T a^4(t)\, dt)^{1/4}$$

Limitations

The new Draft Revision appears to be an improvement over the existing ISO 2631 standard since it incorporates a wider variety of exposures. The decision to remove limits from the main body of the standards is not acceptable to all researchers. There is also resistance to eliminating the rms measure since it makes comparison with past research difficult. There is a lack of evidence to substantiate the new weighting curves and the fourth power analysis (Boileau, 1988). For some vibration environments, the new draft is stricter by a factor of four (Boileau, 1988). The concept of an accumulating vibration dose measure for health effects is an improvement; however, it does not characterize the temporal nature of impacts, which may be linked to long term health effects. Assessment of the effect of vibration on health is made independently along each axis.

British Standard 6841 Measurement and Evaluation of Human Exposure to Whole-Body Mechanical Vibration and Repeated Shock 1987

The philosophy of the new British Standard is to improve methods shown to give erroneous guidance, simplify methods that are unnecessarily complex, and extend the scope of standards to new situations and conditions (BS 6841, 1987). The main function is to provide superior measurement procedures for evaluating vibration rather than to determine limits of exposure (British Standards Institution, 1987). Due to lack of consensus within the ISO TC 108 committee, the British Standards decided to republish 6841 in 1987.

Computational Method

Like the TC108/SC4 draft, this standard is applicable to motions transmitted to the human body through the buttocks, of a seated person, feet of a standing person and the supporting area of a recumbent person. It also applied to x-axis motions of a backrest to a seat. Frequency weightings are applied to the appropriate accelerometer signals in the same manner as in the latest Committee Draft Revision of ISO 2631. In this case, 5 different weightings, Wb, Wc, Wd, We, and Wg are specified for the frequency range 0.5 to 100 Hz These weightings correspond to different axes and applications, namely z-seat (wb), x-back (wc), x and y-seat (wd), rotational x, y, z (we), and z-seat (wg for hand control and vision). A sixth frequency weighting (wf) is specified for the z component in the frequency range 0.1 to 0.5 Hz, for assessing motion sickness. Band-limiting filters are also specified to remove signals outside the frequency range of interest. The band-limiting and frequency weighting are presented as realizable filter characteristics (i.e. magnitude and phase) which can be implemented in analog or digital form.

Evaluation of vibration and repeated shock with respect to health effects

For vibration exposures of constant magnitude and with crest-factors of less than 6.0, the frequency weighted rms acceleration is determined in the x, y and z-axes at the seat and in the x-axis at the seat backrest.

When either crest-factors exceed 6.0 or vibrations are of a variable magnitude, the preferred method for assessing the effect on health is to calculate the vibration dose value (VDV). This method is described in Appendix A of the standard. The dose measure is the fourth root of the integral of the weighted acceleration raised to the fourth power, the integral being taken over the total period during which the vibration occurs:

$$VDV = \left(\int_0^T a(t)^4 dt\right)^{1/4}$$

For cases when the crest-factor is less than 6.0, an estimated vibration dose, eVDV, may, alternatively be determined:

$$eVDV = [(1.4a)^4 \cdot b]^{1/4}$$

where: eVDV is the estimated vibration dose value; a is the rms acceleration in $m.s^{-2}$; and b is the duration in seconds.

For vibration exposure in more than one axis, the fourth root of the sum of the fourth powers of the VDV in each axis is determined to give the total vibration dose value for the environment. It should be noted that calculation of VDV's as

described in Appendix A of the standard are said to be for information only, and are not part of the guide.

This standard does not contain limits for health effects but in the Appendix, a VDV of 15.0 is said to cause severe discomfort (British Standards Institution, 1987).

Limitations

The limitations of this standard are similar to those for the 1991 Draft Revision of ISO 2631. Additionally, the VDV dose measure does not fully account for the effect of repeated cycles, such as in material fatigue, that may be a factor in long term health effects. For example, two sinusoidal signals with identical amplitude and duration, but different numbers of cycles will have similar VDVs. If damage to the body, as is the case for material fatigue, is a function of the number of cycles, the VDV will not appropriately characterize this.

Verin Deutscher Ingenieure (VDI 2057, 1986)

Since 1963, the K value, a dimensionless quantity, has been used in the Federal Republic of Germany to characterize vibration stress (Dupuis and Zerlett, 1986). The K value was based on physiological, subjective and physical studies, many of which were conducted by Dieckmann in the 1950s (Griffin, 1990). The K value was initially designated as "perception quantity", but since 1981 has been called the frequency-weighted vibration quantity. The latest version of the VDI standard (2057, 1986) uses the frequency contours defined in ISO 2631 (1974) to define the K value. For the z-axis of sitting or standing persons, the rms acceleration is converted to KZ values where:

$$KZ = 10\ a_w.f^{1/2} \qquad 1 \le f \le 4$$

$$KZ = 20\ a_w \qquad 4 \le f \le 8$$

$$KZ = 160\ a_w/f \qquad 8 \le f \le 80$$

For x and y-axes:

$$KX = KY = 28a_w \qquad 1 \le f \le 2$$

$$KX = KY = 56\ a_w/f \qquad 2 \le f \le 80$$

where: f = frequency in Hz

a_w = weighted acceleration

The evaluation of the K value (or a_w) is based on guidelines for comfort, performance and health. The simple classification

used is: not perceptible (<0.1), just perceptible (0.1 to 0.4), easily perceptible (0.4 to 1.6), strongly perceptible (1.6 to 6.3), very strongly perceptible (6.3 to 100, or more).

Examples of vehicle accelerations measured using the K value are presented in Appendix B, Tables B-14 to B-17. Typical levels (KZ) for automobiles are 4 to 15, for tractors are 8 to 25 and for military vehicles are 26 to 80.

Dynamic Response Index (DRI)

The Dynamic Response Index (DRI) was developed to characterize the severity of vertical shocks and potential for spinal injury resulting from aircraft ejections (Payne, 1978; Payne, 1968; Payne, 1975). The DRI is based on a single-degree-of-freedom biodynamic model of the spine and upper body. The model consists of a simple spring, mass and damper. The undamped natural frequency, w, is 52.9 rad./second, and the damping ratio, c, is 0.224. Input to this model is the z-axis input acceleration, i.e. seat-pad acceleration. The DRI was originally defined as the ratio of the peak force in its spring to the mass of its model, which has the units of acceleration. This was then made a dimensionless number by dividing by the acceleration of gravity (g). Thus:

DRI = Peak force in the spring/Mass of the model * g

The DRI method firstly determines the net deflection of the mass-spring-damper model, (using the complex transfer function of the model). The DRI is then related to the peak value of the spring deflection, due to the input acceleration wave form, in the following equation:

$$DRI = \omega^2 \delta / g$$

ω = undamped natural frequency (rad./sec)

g = acceleration of gravity

δ = maximum deflection of model

Note that this results in the DRI being a non-dimensional number. The DRI can be interpreted as the maximum equivalent static acceleration in g-units produced by a shock or oscillation. In addition, as formulated, an unaccelerated subject possesses a DRI of 0, rather than the normal 1.0 g value.

Ejection seat data is used to calibrate the injury rate based on the calculated DRI. A DRI over 24 will give a 50% injury rate while for a DRI below 12 the injury rate is less than 2%.

Limitations

The simple compression model of the spine and upper body probably does not adequately describe the real physical situation, which may include large bending stresses induced by rocking motions of the spine (Sandover, 1982). The model applies only to the vertical or z-axis. The DRI only considers positive shocks i.e. compression loading; and does not consider loading of the spine in tension. In addition, the method does not account for complicated time histories or variations in the rate of application of the acceleration (jerk). A slow rate of loading can produce the same DRI as a sudden shock. The DRI model attenuates high frequency vibrations since they are not considered important in spinal injury (rapid attenuation at frequencies higher than 10 Hz). This results in excessive allowable levels at high frequencies compared with curves developed from subjective data (Griffin, 1990). The original DRI did not account for repeated impacts. No information is available about the effects of lower levels of impact occurring repeatedly.

Air Standards Coordinating Committee (ASCC 1982)

The Air Standards Coordinating Committee developed a guideline to give provisional guidance regarding criteria for exposure of aircraft crew-members and ground support personnel to repeated shocks (Air Standardization Coordinating Committee, 1982). It is based upon work by Allen and Payne (Allen, 1977). It was surprising that only three years after the ISO 2631 was published, a public plea was made and an urgency stated for more data on shock and development of better standards (Allen, 1977). Allen and Payne's guideline uses the DRI procedure and takes into account the peaks of repeated impacts by arranging them into an exceedance format, similar to that used to calculate cumulative fatigue damage in metals (Miner's Rule) (Miner, 1945). Proposed criteria are provided in the form of curves. One family of three curves plots the DRI as a function of the number of shocks per day, thus appropriate for assessing repeated daily exposures. The upper curve defines high amplitude shocks requiring 100-day recovery. The injury limit and severe discomfort line are based on a small amount of data from seat ejections, Mirage aircraft, and German tank crew maneuvers. Healthy men in aerobatic maneuvers sustained DRIs of 5 or 6 up to 100 times per day with few, if any, reports of spinal injury (Allen, 1977).

Limitations

The ASCC guidelines are concerned only with accelerations in the vertical direction, and assume that damage is a linear function of accumulated loads. For example, in the case of buckling of the spine or for large loads, damage may accelerate in a way analogous to the behavior of a metal after it reaches

yield point. In addition the standard applies only to seated healthy men, is based on a small amount of data, and the long term effects of repeated impacts, especially of a lower level (DRIs less than 1), are not addressed. The effects on soft tissue may be important in this range, and certainly affect driver discomfort (Forshaw and Ries, 1986).

Hazard Dose Value (Griffin, 1982)

Prior to the publication of the British Standards (6841: 1987), Griffin recommended the use of a Hazard Dose Value (HDV) as a method that gives reasonable indications of the severity of isolated shocks (Griffin, 1982). It is similar to the curves recommended by Allen and Payne (derived from the DRI). The HDV is defined as,

$$HDV = \frac{1}{60}\int_0^T a^4(t)dt$$

Apart from a single publication, little information could be found about the HDV. Its use seems to have been discontinued in favour of the VDV of the British Standards 6841 (1987).

Japanese developments(Kanda et al 1982)

Kanda et al (1982) reported radiological disorders of the spine among crew members on high speed boats due to repeated shocks. The authors developed an exposure limit from their data.

Computational Methods

The Dynamic Response Index (DRI) was applied to the measured vertical accelerations on the seats and wheel house floor of various high speed craft. The vertical acceleration from the seatpan is first passed through an algorithm which transforms it to a DRI vs time record. The number of occurrences of positive (compression) DRI peaks are counted for 100-second records. Peaks less than 0.5 DRI are discarded since they are not considered significant (no positive proof of this was given). Peaks are presented in ranges from 0.5 to 0.9, 1.0 to 1.9, 2.0 to 2.9 DRI etc. DRIs exceeding 5.0 were not observed.

The authors set a tentative daily exposure limit of DRI exposure based on preventing the occurrence of what they called "great vertebral deformation" observed in their study (Kanda et al. 1982). The equation is:

$$\log DRI = 0.5 \log N + \log DRI \cdot N^{1/2}$$

where: N = number of occurrences of the DRI per day larger than 0.5

Also given is an equation which calculates permissible exposure time:

$$T = 1.440/(\text{SUM}[(DRIi)^2 Ni])$$

where: T = permissible exposure time/day

Ni = number of occurrences per hour

DRIi= average of i-th DRI range (i.e. if range is 1.0 to 1.9 then DRIi = 1.5).

Limitations

It is not entirely clear from the study how the authors derived the limit curve. It is presumed to be synthesized from spinal disorder data observed among crew members. In the study, the exposure of crew to high speed ship impacts was only three years, therefore the curves may not be sufficiently conservative for longer exposures.

Other Methods Using Biodynamic Models

Various biodynamic approaches have been proposed to model the behavior of certain parts of the human body under the influence of vibration. Payne has suggested a spinal model, a visceral model, and a whole-body model(Payne, 1978). These models each have different natural frequencies and transfer function characteristics which mimic the modes of their named counterpart. It should be noted that these models are for vertical vibration only.

Fairley and Griffin (1989) determined the apparent mass of 60 seated people. From this data base they developed an average biodynamic model which is based on a single-degree-of freedom system with a natural frequency of 5 Hz and damping ratio of 0.475.

Limitations

It should be recognized that any biodynamic model is only a crude approximation to the real situation and is often only used for computational purposes (i.e. processing input data). Most of these models are passive analogs, which in no way mimic the actual adaptive, active systems within the human body. In the future, more sophisticated models of the body, which include the

effects of active muscle adaptation, may need to be formulated, to model more accurately the body's response to vibration. Additionally, few models have been validated with health effects.

Janeway's Criteria

Computational Method

Janeway (1975) has proposed comfort limits for exposure to vertical vibration. Janeway divides the frequency spectrum from 1 to 60 Hz into three regions. In the first region (frequency range from 1 to 6 Hz) Janeway postulates that "jerk" dominates the comfort criteria. Data predominantly from elevator designers was used. Janeway's recommended limit in this range is : $af^3=2.0$, where a is the motion amplitude in inches and f is the frequency in Hz, corresponding to a constant jerk of 41 ft/sec^3. In the second region (6 to 20 Hz), the limit is: $af^2 = 1/3$, corresponding to a constant acceleration of 0.33 g's. In the third region (20 to 60 Hz), the limit is : $af = 1/60$, corresponding to a peak velocity of 1.0 in/sec.

Limitations

Comfort criteria may be important for health considerations, since as we move from comfortable to uncomfortable situations, we are being made aware of certain possible detrimental effects by our sensory systems. Unfortunately, Janeway was only considering continuous vertical vibration when he set his tolerance limit criteria. These results may not apply to random vibration with repeated impacts.

Absorbed Power

Absorbed power was first considered by Coermann in 1963 and later developed by Pradko and Lee (Pradko, Orr, and Lee, 1963; Pradko, 1966). It is a measure of the average rate at which energy is dissipated by the body as a result of the complex damping mechanisms within the human body. In demonstrating the value of the absorbed power method, Pradko verified that there was a strong correlation between absorbed power and subjective evaluation. In these particular studies, it was not clear if there were repeated impacts contained within the vibration signatures.

Computational method

In the time domain the average absorbed power can be measured directly, i.e., by measuring force and velocity input to the body. The average absorbed power is the integral over time of the product of force and velocity divided by the total time over which the measurement is taken.

Conversely, the absorbed power can be calculated using frequency domain techniques once the frequency response characteristics of the human body are known. The calculation is then as follows:

$$\text{Absorbed Power} = (K_i A_i^2)$$

where: A_i^2 = Mean square acceleration at frequency i

and K_i = Coefficient related to frequency response at frequency i.

This calculation can be performed for all three, x, y, and z translational axes and the output is normally calculated in watts (Norswothy, 1985). Absorbed power of 6 to 10 watts in off-road vehicles is considered the upper limit of what an operator will tolerate for 4 to 6 hours (Pradko, Orr, and Lee, 1963; Pradko, 1966).

The absorbed power method has appeal, since it has the ability to reduce the oscillatory components of ride to a single scalar quantity. This method is used as a measure of ride quality for evaluation of military vehicles by the U.S. Army Tank and Automotive Command (TACOM) and the Geotechnical Group at the W.E.S. Laboratories in Vicksburg, Mississippi (Willoughby, 1981; Gillespie, 1984; Murphy and Ahmad, 1986; Murphy, 1984; Nuttall et al. 1985).

Limitations

Norsworthy (1985) compared absorbed power to other methods of analysis for ability to order the severity of rides. The correlation between subjective measure and absorbed power level was low when compared with some of the other measures such as ISO rms. It may be that Norsworthy's (1985) study was looking at comparatively low absorbed power levels (1-2 watts). Non-linearities in the damping behavior of the body may explain the results and thus may need to be incorporated into the calculation procedure.

Von Gierke (1975) points out that the absorbed power does not adequately correlate with human response at frequencies below 1 Hz. The absorbed power model predicts increasing tolerance, while the adverse effects of motion sickness at frequencies below 1 Hz are well known.

Since the absorbed power is an averaging procedure performed over a frequency range, its sensitivity to transients and peaks is questionable. It was not evident in Pradko's paper if transients or jolts were present since the vibration was described as random, characterized by a single rms value.

USSR Methods

Menshov (1979), briefly describes the principles for rating whole-body vibration in the USSR. The rating is based on vibration velocity in octave bands, rather than acceleration in one-third-octave bands. The use of vibration velocity rather than acceleration to quantify vibration effects is reported to be validated through physiological indices (Menshov, 1979).

Limitations

The data presented in this paper is sparse and no other studies have been found that support the use of velocity as a method of characterizing human response to vibration. Historically, acceleration has been easier to measure than velocity when studying vibration, although this is not a problem today.

Summary

The most recognized standard for human response to whole-body vibration is the International Standards Organization (ISO) 2631. However, lack of consensus among members of the technical committee (TC108/SC4) has impeded publication of a major revision since 1982. Recent ISO Draft Revisions appear similar in many respects to the British Standards (6841: 1987). These new standards incorporate rmq and vibration dose values (VDV) as the main method of characterizing vibration with shocks and repeated impacts. Limits of over-exposure have been removed from the body of the standards and placed in Appendices as guidelines. Both the ISO Draft Revision and the BS 6841 state that epidemiological evidence supports the 4 to 8 hour vertical acceleration limit of the previous ISO 2631 (1982) Standard, and that this is roughly equivalent to a VDV of 15. Little evidence exists to support use of these guidelines for signals with repeated shocks of high magnitude.

Two guidelines were found for exposure to repeated impacts, both using the dynamic response index (DRI) as their basis (Air Standardization Coordinating Committee, 1982; Kanda et al. 1982). The Air Standardization Coordinating Committee curves of severe discomfort plot the number of shocks in 24 hours as a function of the DRI. A different curve plots the 5% injury probability over a

100 day recovery. Kanda's (1982) tentative daily exposure is based on a study of spinal disorders among crew members of high speed ships. It is not clear from the study how the authors derived the limit curve. Although validated with health data, both guidelines are limited to models of spinal injury and to repeated impact in the vertical axis. No standards consider recovery explicitly in their models.

Vibration data collected in the field

Introduction

There is an abundance of reports and studies that have sought to determine the level of vibration in a variety of situations, including on-road vehicles (cars, trucks, buses), off-road vehicles (tractors, skidders, tanks, construction equipment), air transport (helicopter, fixed wing), water transport (ship, hovercraft, hydrofoil), and a variety of miscellaneous vehicles (motorcycles, amusement park rides, bicycles, subways). The purpose is often to compare the level of vibration with some standard (usually the ISO 2631 guideline) and determine if vibration exposure is excessive. Sometimes the purpose is to compare vibration exposure between various situations (such as good road surfaces and poor ones), and at other times the purpose is to evaluate the seating or suspension of a vehicle. This section provides a summary of vibration data that have been collected for various purposes. Details are presented in a series of tables and brief descriptions of the trends are provided.

Despite the efforts of the International Organization for Standardization (ISO 2631) to specify measurement and analysis techniques, not all authors have followed the standardized guidelines. Hence, a variety of ways of analyzing and presenting data exist in the literature. Less attention was given to unstandardized methods, since this data cannot be readily compared with that of other authors. The vast majority of measurement has been made in the vertical (z) axis, with approximately one-third of the reports also providing horizontal (x and y) axes measures. Unfortunately, due to the option provided in ISO 2631 (1978) of reporting either the dominant one-third octave, or the weighted vibration (rms) from the full spectrum, it is not always clear from the methods in the studies which option was chosen. Few papers (approximately 20%) reported crest-factors or vector sums. Some of the more recent papers have included vibration dose values (according to the British Standards 6841), and many of the German papers have included K-values. Where provided, the dominant third-octave frequency bands are reported.

Summaries of the data are provided in Appendix B, Tables B-14 to B-17. Table B-14 lists vibration levels in on-road wheeled vehicles; Table B-15 lists off-road vehicles; Table B-16 lists Aircraft and Table B-17 lists miscellaneous vehicles. The variety of data-reporting methods made the formation of summary tables difficult. Some authors reported the number of measurements or the percentage of vehicles that exceeded the ISO exposure guidelines, rather than reporting acceleration levels. There was no way to estimate the mean acceleration levels from this information. Other authors provided the equivalent exposure time

according to the ISO boundaries. Exposure times were converted to the appropriate acceleration levels. In some papers, results were presented graphically, but actual levels of acceleration were not given. Where levels have been estimated from graphs, or converted from exposure times, a note is made on our summary tables. Data were only accepted for incorporation into these tables if weighted and analyzed according to ISO 2631.

A surprising range of acceleration values is found between authors for the same type of vehicle. There are a number of reasons why this might occur. Measures will vary according to the measurement procedure (such as length of measurement period), type of terrain over which the measures were taken, speed of travel, experience of the driver, instructions given to the driver, whether the equipment was loaded or unloaded, and the total number of measures taken. Some experimenters were looking for the worst case exposure, others were looking for typical levels. A wide range of values is often provided by individual authors to represent the variation in operational vibration levels for a particular vehicle. For example, idling vibration levels will be much lower than those encountered during peak work periods. With such a wide range, it is difficult to determine what a typical exposure is for an equipment type.

Wheeled Vehicles

Cars and buses tend to have lower vibration and shock levels compared with trucks and ambulances, especially if driven on paved surfaces (Dupuis, 1980) (Appendix B, Table B-14). Levels of acceleration in the z-axis for cars on a variety of surfaces ranged from 0.2 to 1.0 $m.s^{-2}$ (Nelson and Lewis, 1989; Dupuis and Zerlett, 1986; Dupuis and Zerlett, 1987; Griffin, 1978; Dupuis, 1980; Dupuis and Zerlett, 1986; Griffin, 1984). Horizontal vibration for cars is reported to be low (a_x = 0.02 to 0.4, a_y = 0.05 to 0.45), as are the crest-factors (approximately 5). The dominant frequencies are reported to be from 6 to 12 Hz in the vertical direction and from 1 to 3 Hz in the horizontal directions.

Dupuis (1986; 1987) found buses to be within the same range of acceleration as cars (0.3 to 0.8 $m.s^{-2}$), but Griffin (1978) reported a higher range (0.5 to 1.75 $m.s^{-2}$). Horizontal acceleration in buses is also low and the dominant frequencies are similar to those of cars. Vans were reported to have a vertical acceleration of 0.37 $m.s^{-2}$ on paved roads and 0.65 $m.s^{-2}$ on country roads (Griffin, 1984).

Ambulances were measured to have vertical vibration from 0.5 to 1.6 $m.s^{-2}$ (Dupuis, 1980). Trucks were similar with a range of 0.4 to 1.5 $m.s^{-2}$ (Griffin, 1978; Dupuis and Zerlett, 1986; Dupuis and Zerlett, 1987; Boulanger, Donati, and Galmiche, 1986; Boshuizen, Bongers, and Hulshof, 1990; Dupuis, 1980; Wilcox

et al. 1988). One study reported a lower range for trucks, from 0.2 to 0.9, probably because they were measured on paved roads (Dupuis and Zerlett, 1986; Dupuis and Zerlett, 1987). While paved roads yield average accelerations of 0.7 $m.s^{-2}$, vibration caused by poor roads usually exceed 1.0 $m.s^{-2}$ (Boulanger, Donati, and Galmiche, 1986). Horizontal vibration is also higher in trucks compared with buses and cars (a_x and a_y = 0.15 to 0.65 $m.s^{-2}$); however, the crest-factors were similar (3 to 9) (Dupuis, 1980). Dupúis reports that trucks on country roads can have crest-factors up to 23 (Dupuis, 1980). Dominant frequencies for trucks are 6 to 12 Hz in the vertical axis (although there is one report of 2 to 8 Hz from Wilcox 1988), and 1 to 4 Hz in the horizontal axes. Most industrial trucks exceed the ISO Fatigue Decreased Proficiency Boundary (FDP) for the typical working day (Boulanger, Donati, and Galmiche, 1986; Hansson, 1977). One study reported subjective assessments of discomfort at the 3-hour limit of the FDP boundary in trucks (Hansson, 1977).

Shock and Vibration in Off-Road, Heavy Equipment

Off-road heavy equipment can also be grouped according to levels with lower levels found in cranes, open-pit mining vehicles, and letourneaus (wheeled forestry equipment used on pavement), intermediate levels in shovels, crawlers, excavators, forklifts, and forwarders; and high levels in dozers, graders, loaders, underground mining (load-haul-dump, LHD) equipment, skidders, tractors, and military tanks (Appendix B, Table B-15).

Cranes, letourneaus and open-pit mining haulage vehicles had vertical vibration in the range of 0.1 to 0.8 $m.s^{-2}$ (Dupuis and Zerlett, 1986; Dupuis and Zerlett, 1987; Village, 1989; Wilcox et al. 1988). Construction-type equipment, including shovels, crawlers, excavators, forklifts and forwarders all had vertical vibration in the 0.3 to 1.6 $m.s^{-2}$ range (although there was evidence for levels up to 2.0 $m.s^{-2}$ in forklifts, and up to 2.5 $m.s^{-2}$ in excavators) (Dupuis, 1980; Dupuis and Zerlett, 1986; Dupuis and Zerlett, 1987; Boulanger, Donati, and Galmiche, 1986; Hansson and Wikstrom, 1981; Mcnulty and Douglas, 1985). Unfortunately, a large U.S. study conducted by Wasserman (1978) on a broad range of construction and other heavy equipment could not be incorporated into the tables since vibration was not analyzed in one-third octave bands, nor weighted according to ISO 2631. Peak accelerations were reported for each single octave.

Dozers, graders, loaders, tractors, skidders and underground load-haul-dump (LHD) vehicles all have vibration in the vertical axis that ranges from 0.5 to 2.5 $m.s^{-2}$ (Monsees et al. 1988; Dupuis, 1980; Dupuis and Zerlett, 1986; Dupuis and Zerlett, 1987; Village and Morrison, 1989). Lower levels were reported by other authors for loaders and dozers (Boshuizen, Bongers, and Hulshof, 1990; Boulanger, Donati, and Galmiche, 1986; Air Standardization Coordinating Committee, 1982). A great deal of study has been

conducted in Canada on skidders which are used in forestry for hauling timber from the bush. Skidders operate in extremely rough, hilly terrain, often travelling over stumps and logs. While the majority of measures fall in the range of 0.6 to 1.4 $m.s^{-2}$, levels up to 2.19 $m.s^{-2}$ have been measured (Golsse, 1989; Hope, 1984; Boileau et al. 1986; Boileau, 1988; Golsse and Hope, 1987; Boileau and Scory, 1986). Vertical vibration in tractors has been measured up to 4.5 $m.s^{-2}$ (Matthews, 1964), and average values have been reported from 0.35 (Boulanger et al. 1978) to 2.8 and 2.9 $m.s^{-2}$ (Matthews, 1964; Dupuis and Zerlett, 1986).

Tractors, skidders and mining load-haul-dump (LHD) vehicles have high vibration in the horizontal axes as well (a_x and a_y = 0.5 to 1.9 $m.s^{-2}$). Crest-factors in skidders have been reported up to 10, in tractors up to 13 and in LHDs up to 20 (Boileau et al. 1986; Boileau, 1988; Clarke et al. 1966; Monsees et al. 1988; Village and Morrison, 1989). Dominant frequencies in these vehicles are very low, from 0.6-2.5 Hz in the vertical direction and from 0.5 to 2.3 Hz in the x and y directions. Boileau (1986) reported that the FDP boundary is exceeded in skidders in at least one direction after an average of 2.7 hours. He found that the vertical acceleration calculated by the overall weighted spectrum overestimate by a factor of three the acceleration analyzed according to the dominant one-third octave value (Boileau et al. 1986). Similarly Morrison, Village and Kent (1991) compared LHD vibration using a number of analytical methods and found that all vehicles were close to the exposure limit (EL) using the dominant one-third octave technique. However, when weighted spectrums were evaluated, all vehicles exceeded the EL. When vector sums were calculated, vehicles exceeded the EL by a factor of 1.4 to 5.4 (Morrison, Village, and Kent, 1991). Mining equipment measured in Sweden was found to have similar levels (Wikstrom, 1979).

The highest levels of vibration found in the literature were for certain types of military vehicles (Griffin, 1984; Bennet, Farmilo, and Campbell, 1974; Dupuis, 1974; Breen and Calderon, 1989; Cole, 1977; Butler and Maday, 1986; Dupuis and Zerlett, 1986; Dupuis and Zerlett, 1987; Fraser, Smiley, and Mottershead, 1976). Unfortunately, the types of vehicles are not identified in many of the papers. The highest levels were from Dupuis (1974) who reports vertical acceleration of 4 to 6.0 $m.s^{-2}$ on concrete roads and rough field tracks and up to 10.0 $m.s^{-2}$ on belgian blocks (Dupuis, 1974). Maximum exposure limits are exceeded in the z-axis, in some cases in as little as half an hour. These are a level of magnitude above later reports of the Canadian armoured fighting vehicles (Cole, 1977; Crabtree, 1982). The FDP boundary was exceeded in 0.5-1 hour depending upon the seat position, terrain, and type of vehicle (for example, the FV 432) (Cole, 1977; Crabtree, 1982). USAARL measurements of the towed vulcan air defense system (TVAD) showed that levels in the x-axis would exceed the FDP boundary in one minute on cross-country terrain (approximately 3.0 $m.s^{-2}$)' however average values ranged from 0.78 to 1.2 $m.s^{-2}$ (Butler and Maday, 1986). Tracked cargo

carriers, the vulcan wheeled carrier, and the M-88-A1 armoured fighting vehicles (AFV) were less severe, with the EL being exceeded on cross-country terrain in the tracked cargo carriers in 3.1 hours, the FDP in the vulcan wheeled carriers (VWC) in 6 hours and most AFV measures falling within the 8-hour FDP boundary (Miller and Breen, 1989; Butler and Maday, 1986; Breen and Calderon, 1989).

Additionally, military vehicles have some of the highest impacts of all vehicles. In a four minute exposure period on the FV 432, there were 4 impacts with DRIs of 2 (Cole, 1977). When an all-terrain vehicle was measured on a heavily plowed track, it was estimated the EL would be exceeded in one-minute (Fraser, Smiley, and Mottershead, 1976). Measurements were not taken in this study according to ISO; however, they reported that acceleration in the z-axis was above 5 $m.s^{-2}$ for 26% of the time and above 20 $m.s^{-2}$ for 0.5% of the time (mostly shocks) (Fraser, Smiley, and Mottershead, 1976). Dupuis mentioned in one report that investigation of military vehicle operators' spines led to a connection between exposure and injury (Dupuis, 1974). However, he gives no details or data in this report to validate this statement.

Griffin (1990) illustrated the effect that travelling speed has on tank vibration when travelling on a test course. When speed was varied from 2.4 to 4.5 $m.s^{-1}$, vertical acceleration went from 1.3 to 2.5 $m.s^{-2}$ (3-hour EL to 1-hour). Also increasing with travel speed were crest-factors, from 3.3 to 14.4 (Griffin, 1990). Clearly, this type of acceleration spectra, with high level shocks, cannot appropriately be analyzed with current ISO standards (Village and Morrison, 1989).

Shock and Vibration in Air Transport

Motion in aircraft is a combination of that from rotating parts within the aircraft and air motion and turbulance outside the aircraft. Levels for helicopters range from 0.05 to 1.5 $m.s^{-2}$ (Griffin, 1984; Griffin, 1975; Bongers et al. 1990; Dupuis and Zerlett, 1986; Dupuis and Zerlett, 1987) (Appendix B, Table B-16). In one study the type of helicopter was differentiated (Griffin, 1984). Levels for transport helicopters were lower in the vertical directions (0.73 $m.s^{-2}$) than for military helicopters (1.14 $m.s^{-2}$) (Griffin, 1984). Most studies have reported similar levels in the horizontal directions to be 0.04 to 1.3 $m.s^{-2}$ (Bongers et al. 1990; Griffin, 1975). Crest-factors in helicopters are low, usually less than 3. Dominant frequencies are higher than ground transport in all directions: 8 to 25 Hz in z-axis, 16 to 25 Hz in x-axis and 6 to 20 Hz in y-axis. Evaluations of two military helicopters (UH-1 and UH-60) revealed 42.7% and 54% of measurements to be within the FDP boundary. Other methods have been devised for evaluating helicopter vibration (Griffin, 1975). In one method, it states that

acceleration in normal flight should not exceed 0.3 $m.s^{-2}$ in the horizontal directions and 0.4 $m.s^{-2}$ in the vertical (Griffin, 1975). The U.S. Army Aviation System Command standard ADS-27 is a statistical measure of mechanically induced rotary-wing aircraft vibration, based on vibration from the aircraft frame and derived from pilot performance data (Hosea et al. 1986).

Fixed wing planes typically have low vertical vibration (0.36 $m.s^{-2}$), but levels up to 1.25 $m.s^{-2}$ were reported for a military fixed wing aircraft (Griffin, 1990). Crest-factors range from 4 to 5.3.

Shock and Vibration in Miscellaneous Environments

At the low end of the acceleration spectrum are vibration measurements in buildings (0.029 $m.s^{-2}$) (Griffin, 1984). Vibration measured in water transport has yielded vertical acceleration levels for ships of 0.2 to 0.7 $m.s^{-2}$, hovercraft of 0.47 $m.s^{-2}$ and hydrofoil of 0.08 $m.s^{-2}$ (Griffin, 1984; Dupuis and Zerlett, 1986; Dupuis and Zerlett, 1987). Vibration in railed vehicles, such as passenger trains, tends to have low vertical acceleration (0.3 to 0.8 $m.s^{-2}$), and very low x-axis vibration (0.05 $m.s^{-2}$), but Heino et al (1978) found fairly high y-axis vibration (0.75 $m.s^{-2}$) compared with Griffin (1978) (0.12 $m.s^{-2}$). Dominant frequencies for trains are 2 to 4 Hz in the z-axis, 2 to 2.5 Hz in the x-axis and 1.25 to 4 Hz in the y-axis. Heino et al (1978) reported exceedences of the 8-hour FDP boundary in 31, 20, and 43%, of x, y and z axes, respectively. Vibration levels for subways were not unlike trains, with vertical levels ranging from 0.27 to 0.69 $m.s^{-2}$, and very low (<0.5 $m.s^{-2}$) motion in the horizontal directions (Johanning et al. 1991).

Measurements have recently been made on motorcycles and all-terrain bicycles on various road conditions. Motorcycles ranged from 0.9 to 1.5 $m.s^{-2}$ in the vertical direction and had low values in the horizontal directions (0.1 to 0.4 $m.s^{-2}$) (Nelson and Lewis, 1989). Bicycles on country tracks measured 1.36 to 2.1 $m.s^{-2}$ in the vertical axis and 0.6 to 1.2 in the horizontal axes, with crest-factors an amazing 30 (Nelson and Lewis, 1989; Lewis and Paddan, 1990). Bicycle riders will anticipate the bumps, and rise up from the seat onto their feet to absorb some of these impacts with their legs.

The highest vibration measurements were found for amusement park rides, some of which were in poor maintenance (Irwin, 1977). The most exhilerating rides had dominant frequencies in the 6 to 8 Hz range and vertical vibration levels of 10 to 40 $m.s^{-2}$. It is clear that people are willing to accept much higher levels of vibration in amusement park situations when they are paying for the ride than in other situations. The author suggested loud music increased voluntary exposure and that there is a gradual

acclimatization as the ride accelerates up to operating speed. Also, most people will only ride once per day, therefore, the exposure is rated at only "moderately uncomfortable" (Irwin, 1977).

Conclusions

Levels of vibration and impact have been collected from a wide database of studies. It is useful to characterize typical levels in the various applications. USAARL data can then be compared with data collected by other researchers. Ultimately, data from these field studies will be combined with a database of epidemiological health effects to attempt to better understand the types of exposures associated with various health problems.

Despite the efforts of the International Organization for Standardization to standardize measurement and analysis of whole-body vibration data, a wide range of methods exist in the literature. Where possible, data from studies was converted into rms measures, and weighted according to the curves in ISO 2631 (1978). Tables were constructed to allow comparison from various researchers in categories of: on-road wheeled vehicles (buses, cars and trucks); off-road heavy equipment (tractors, bulldozers, skidders and fighting vehicles); aircraft (helicopters and fixed wing); and miscellaneous vehicles (ships, motorcycles, subways and trains).

Some of the highest levels of vertical acceleration and crest-factors were reported for military vehicles. A large range in acceleration levels reflects different measurement procedures, types of terrain, speed of travel, experience of driver, instructions given to the driver, whether the equipment is loaded or unloaded and the total number of measurements taken.

Signal Processing

Vibration consists of continuous or transient oscillations that can be quantified using displacement, velocity or acceleration measurements. Most work in human vibration is performed by taking acceleration measurements of the vibration input to the human body.

Acceleration signals may be deterministic or random. Deterministic vibration may occur as periodic motion (e.g. sinusoidal) or non-periodic motion (e.g. transient, or impulsive). With deterministic vibration, a unique phase value is associated with each frequency component. This implies that the future behavior of the signal can be accurately predicted knowing its past. With random vibration, the phase of each frequency component is random and future values of the signal can only be predicted in a probabilistic manner. Random vibration may be stationary, where the root-mean-square (rms) acceleration level does not vary with time, or non-stationary, where rms varies with time. Impact has been defined as a single collision of one mass with another mass (Griffin, 1990). The transition from impacts, to shocks, to repetitive shocks, to bumps, to vibration, is arbitrary (Griffin, 1990).

In practice, combinations of the above frequently occur, and this leads to difficulties in establishing appropriate criteria to describe human responses and health effects. Of primary concern in this study is the specification of human response to vibration containing mechanical shocks and impacts. Shock is used in this review to describe a mechanical input to the human body (force, displacement, velocity and acceleration) that changes in a time duration shorter than the natural response time of the body, which is in the range 100 to 200 ms. Thus, shocks cause rapid, forced disturbances in the relative positions of body parts and may result in excessive peak stresses within tissues, ligaments or bones.

The role of signal processing in this project is to use mathematical methods to quantify the complex acceleration wave forms to allow their effect on the human body to be assessed. The treatment explores analysis in the frequency domain, the time domain and combinations that construct a vibration dose. A multi-disciplinary approach is taken which combine current knowledge of the response of the human body to vibration and shock with signal processing and analysis techniques which in our opinion are the most useful.

Data Acquisition

In the past, researchers have had to rely on analog recording and analysis systems. The computing power and data storage

capabilities of modern digital computers provide much more freedom in data acquisition and analysis and effectively remove the limitations of analog systems. When working with data in digital form, however, there are certain theoretical considerations and technical limitations that the researcher must be aware of to avoid problems when interpreting analyzed results.

Some considerations when acquiring digital vibration data are; the data sample rate, record length, and the lowest and highest frequencies contained in the time history. When performing spectral analysis these factors influence the frequency resolution and accuracy of the spectral estimate.

Procedures for estimating sample rate and record length have been detailed in the literature (Butz, 1979; Bendat, 1958; Newland, 1975; Bendat and Piersol, 1966). Sample rate is an important factor which can drastically affect the spectrum and the representation of high-frequency components in the wave form. The problem of aliasing (the appearance of high frequency components at a lower frequency) can be prevented by sampling at a frequency at least twice as high as the highest frequency component in the signal being analyzed. Selection of suitable analog anti-aliasing filters of the raw signal can control this problem. For example, when recording data, anti-aliasing filters with a cut-off frequency of 500 Hz should be used. A sample rate of 2000 Hz will then avoid aliasing. The high sample rate is also employed to define, precisely, peak amplitudes at all relevant frequencies (1990).

Low frequency resolution can be adversely affected by time records which are too short in duration. The time series record must be long enough to contain at least one full cycle of the lowest desired frequency. For example, to analyze signals containing a frequency of 0.1 Hz, the minimum time record is 10 seconds.

Frequency Domain Analysis

Historically, the majority of human vibration analysis has been carried out in the frequency domain. This early emphasis was probably encouraged by the available (analog) instrumentation which could readily quantify the effects of vibration on humans at different frequencies. At that time the methods for quantifying vibration magnitude were comparatively primitive.

Using the basic assumption of linearity, knowledge of the system transfer function, and the input excitation spectra, the resulting response of a linear system can be determined. Powerful methods, (such as Fast Fourier Transform and various data windowing techniques) have been developed to reduce complex vibration characteristics to relatively simple response spectra (Butz, 1979; Bendat, 1958; Newland, 1975; Bendat and Piersol,

1966; Blackman and Tukey, 1959). Much can be learned, such as natural frequencies, vibrational modes and damping behavior, by studying the characteristics of these spectra.

Stationarity

Central to any discussion of spectral analysis is the concept of stationarity (Newland, 1975; Bendat and Piersol, 1966). The basic definition of stationarity is that the mean, standard deviation and all higher order statistics of a time series do not vary significantly from one record within the time series to the next. When using a spectrum to characterize a given vibration environment, it is necessary that the condition of stationarity apply. If this is not the case, the spectrum computed from one time interval record will not correspond to that computed from some later time interval.

In practice, most data are non-stationary. Time records are divided into quasi-stationary records to perform spectral analysis. The evolution of the spectrum over time can then be plotted graphically showing any non-stationary behavior. This is the recommended procedure for continuous vibration (Bendat and Piersol, 1966). The analysis of vibration containing impulsive signals and jolts is another matter, and is the subject of much debate. By their very nature, impulses and jolts are non-stationary, and therefore are not amenable to standard spectral techniques. For this reason, time domain techniques have been developed for the analysis of signals containing impacts.

Data windowing techniques

A problem associated with short time records is the phenomenon of leakage. This occurs because of abrupt signal level changes at the beginning and end of a time series record if it begins or ends with a non-zero (vibration) value. To prevent these end effects from influencing the spectral estimate, various data windowing techniques can be applied, (Burgess, 1975; Butz, 1979; Bendat, 1958; Newland, 1975; Miles and Funke, 1989). In general, the data window attenuates the portions of the data record at either end. Spurious side-lobes which may appear in the frequency domain, or spectral estimate are thereby reduced. In most frequency domain data analysis, the Hanning data window is used (Burgess, 1975).

Fast Fourier transform methods

Many methods and algorithms (such as the Fast Fourier Transform) have been developed over the years for determining the spectral estimate of a time series (Burgess, 1975; Bendat, 1958; Bendat and Piersol, 1966).

The Fast Fourier Transform (FFT), developed by Blackman and Tukey (1959), has been used for many years as an efficient algorithm for calculating spectra. Input to the algorithm is simply the time series (i.e. digitized acceleration record) together with its sample rate. Output are the acceleration amplitudes and phases in the frequency domain. This method is central to the digital 1/3 octave analysis techniques as they are applied in the vibration standards.

An Inverse Fast Fourier Transform (IFFT) technique (Miles and Funke, 1989) is also available whereby amplitude and phase information in the frequency domain is transformed back to the time domain to reconstruct the time series signal. The IFFT, used in conjunction with the FFT, can be used to perform useful filtering and weighting functions.

One of the problems in analyzing vibration signals with imbedded impacts is that the impacts are not well characterized in the frequency domain. An impact has a spectrum with a continuous distribution of relatively small amplitude components. Thus, the spectrum of an acceleration record without impacts may not look much different than one with impacts, even though the impacts may have serious consequences to human health.

Other Methods of Spectral Estimation

SIWEH method

Papers by Funke and Mansard (1980), Burcharth (1979) and Goda (1976) detail a method of wave form analysis called Smoothed Instantaneous Wave Energy History (SIWEH). This method was first applied to the study of the response of offshore structures to waves. The rationale behind this method is that structures, which are subject to random excitation or vibration, respond not only to individual amplitudes (in a manner characteristically determined by inertial and damping properties), but also to groups of successive amplitudes (or wave groups). These wave groups can be quantified and analyzed using SIWEH analysis techniques. The SIWEH is a function of time which describes the distribution of energy of the excitation along the time axis. The spectral density of the SIWEH can be calculated. This analysis generally results in a spectrum which has its frequency peak at least an order of magnitude lower than the input wave signal spectral peak, providing the possibility of eliciting a response at these much lower frequencies.

This method may be relevant and useful when synthesizing vibration records for the three-axis shaker table. Attention may have to be paid to the group behavior (in time) of the acceleration amplitude records in a way which is analogous to the synthesis of wave records, using SIWEH analysis methods.

Shock spectrum methods

Shock spectrum methods have been used by engineers in the past to investigate the response of structures to impacts and transient shocks (Kelly and Richman, 1969; Strong, 1985; Kittelsen, 1966). Harris and Crede (1976) give a complete description of these methods. The shock spectrum can be thought of as the maximum response to a shock impulse of a large number of un-damped, parallel, single-degree-of-freedom mass-spring systems. An analysis of the resulting shock spectra of different types and sizes of shocks reveals that a shock's shape, peak value, duration and area under the time curve are important factors when assessing the ability of the shock to interact with a structure.

Payne (1978), took the shock spectrum concept and developed a simplified model of the human body which was composed of three parallel, single-degree-of-freedom mass-spring-damper systems to evaluate the effects of shocks on the human body. This model, particularly in the version known as the dynamic response index, has found some success in evaluating the human response to seat ejections from aircraft.

Cepstrum methods

One method of deriving information from a signal is calculation of the cepstrum (Flanagan, 1972). The technique is used in speech analysis, and is useful in examining signal components that are widely spaced in frequency. In speech, the vibration of the vocal cords results in a relatively high frequency carrier vibration (500 to 5000 Hz), while the sounding of the vowels occurs with a much reduced frequency (1-250 Hz). To calculate the cepstrum, a suitable (Hamming) window is first applied to a short time record of the speech. The Fourier transform is then calculated, followed by the logarithm of the magnitude of this transform. The inverse Fourier transform of this function is the cepstrum.

The cepstrum is a function of time. High frequency components of the log-magnitude spectrum can be removed by low-pass filtering the cepstrum, and performing the inverse Fourier transform back to the log-magnitude spectrum. Peaks in this resulting spectrum indicate formant (resonant) frequencies that are present.

Although this technique is valuable for analyzing signals with widely varying frequency components, we have yet to determine if it has relevance to our application. This is because we are working with a band of input frequencies (1.0-100 Hz), which is, in the applications we are considering, further narrowed to a range from 1 to about 20 Hz by the filtering effect of the human body. Also, through the application of the FFT in calculating the

cepstrum, the influence of sharp impacts and peaks in the original time series signal will likely be reduced.

Time-Frequency Representations

Several advanced time-frequency formulations have been examined which belong to the general category of joint time-frequency distributions. Of these distributions the most useful is the Wigner-Ville distribution (Sibul, 1991; Meng and Qu, 1991; Marinovc and Eichman, 1985; Claasen and Mecklenbrauker, 1980; Claasen and Mecklenbrauker, 1980; Zielinski, 1989; Arai and Miura, 1987; Boashash and Whitehouse, 1987). The method is somewhat analogous to the spectrograms used in speech analysis. It can display the behavior of a highly non-stationary wave form graphically. The energy content of a signal is displayed on both the time axis and the frequency-axis, resulting in a visually informative three-dimensional plot. Besides being computationally intense, with much redundant information generated, it is considered that these time-frequency representations may not adequately represent or characterize signals containing repeated dissimilar impacts or pulses. The reason for this is that the structure of the acceleration peaks will be lost or blurred through application of the Fast Fourier Transform (FFT) which is used in its calculation. It is envisioned, however, that such time-frequency representations will some day supersede the standard spectral density representation for continuous vibration cases of low crest-factor. This is because the Wigner-Ville distribution can better represent cases where significant non-stationarities exist.

Amplitude-frequency methods

Stikeleather (1975), and Park and Wambold (1985) report a method of analysis known as the amplitude-frequency distribution (AFD) technique. In this procedure, amplitude discriminators (windows) are applied to the output of narrow band filters covering the frequencies of interest. The result is a three-dimensional surface indicating the number of amplitude peaks occurring in each frequency and amplitude band. This method may be used to quantify ride severity by taking a measure of the constructed function, such as its volume. It should be noted that this function preserves both amplitude and frequency information and can be applied to non-stationary data.

Time Domain Analysis

Time domain analysis methods do not suffer the limitations of frequency domain methods for characterizing impacts that vary in magnitude, frequency content and frequency of occurrence. The

advantage to working with data in the time domain is that temporal relationships (including phase) may be preserved. This allows for accurate evaluation and analysis of peak values, derivatives and transients which are present in vibration records.

Various time domain magnitude estimates of vibration signals include rms, rmq (root-mean-quad), and other higher power representations. The classical technique of performing time domain analysis is to determine the probability distribution of the magnitudes of the time series, in particular, the probability density distribution, and derive from it the above mentioned magnitude estimates (rms etc.). In this section, methods for estimating the magnitude of random vibration, with and without shocks, are described. The methods depend on the underlying variation in signal amplitude, which may be described statistically without reference to the passage of time. These methods are well documented in the literature, (Piersol, 1963; Alfredson and Mathew, 1985; Sherf, Yehuda, and Katz, 1983; Xistris, Boast, and Sankar, 1980; Thrall and Bendat, 1965).

The magnitude of random vibration may be described in terms of the properties of its constituent distribution, which is a Gaussian or normal distribution. The expected value, $E(a^n)$ of the probability density function, $p(a)$ is defined, generally, in terms of its second, and higher, order moments. For the second order term (the mean-squared acceleration):

$$E(a^2) = \int_{-\infty}^{+\infty} a^2 p(a)\, da \qquad (1)$$

where:

a = instantaneous acceleration.

and for the n-th order moment:

$$E(a^n) = \int_{-\infty}^{+\infty} a^n p(a)\, da \qquad (2)$$

In general, if the root mean value for the m-th=2n-th order moment is defined from the corresponding expected value by forming the m^{th} root, where m=2,4,6,...etc.

$$[E(a^m)]^{1/m} = [\int_{-\infty}^{+\infty} a^m p(a)da]^{1/m} \qquad (3)$$

Then the relationship between the m^{th} root mean value and the root mean square value is:

$$\frac{[E(a^m)]^{1/m}}{a_{rms}} = [1..3..(m-1)]^{1/m} \qquad (4)$$

For a Gaussian distribution, a probability may be associated with each mean value. These are listed in Table 1, and indicate that the (root-mean-ten), a_{rmd}, value corresponds to P(a)=0.95 in the (cumulative) probability distribution.

Table 1:

Relationships between even order moments and rms value

Moment & Root order, m	Root Mean Value	Relation to RMS	Cumulative Probability P(a)
2	a_{RMS}	1.00	0.68
4	a_{RMQ}	1.32	0.81
6	a_{RMX}	1.57	0.88
8	a_{RMO}	1.79	0.93
10	a_{RMD}	1.98	0.95

Note that all odd-powered moments of a Gaussian distribution with zero mean value are identically equal to zero. The existence of shocks within a (random) vibration will cause the time series to deviate from a Gaussian probability distribution, when the shocks exceed the amplitude variations of the background vibration. The presence of shocks in an otherwise random signal can therefore be established from the shapes of the probability density or (cumulative) probability distributions. Such changes in the shapes of the distributions will be reflected in the relative magnitudes of the expected values, which will deviate from the ratios listed for the corresponding root mean values in Table 1. The cumulative probability, P(a), associated with each mean value (column 4, Table 1) will also change to reflect changes in the amplitude distributions.

Deviation of the values from their Gaussian values are an indication that shocks may be present in the signal. The extent to which shocks are present in the signal will be indicated by examining the ratios of the higher-order means to the r.m.s. and comparing them to the Gaussian values of these ratios.

One problem in common with all distribution-based methods of analysis is that although the vibration amplitudes are completely characterized, their temporal occurrence is not.

Impulsiveness and peak values

An alternative measure of the magnitude of vibration that retains a specific probability value may be obtained by calculating the impulsiveness of a wave form. Stark and Pyykko(1986), report the importance of the impulsiveness factor (peak/rms) in determining the severity to human health of impacts and vibrations.

This factor, or Impulsiveness, $I_{P(a)}$, measure may be defined as:

$$I_{P(a)} = \frac{[a^+ - a^-]}{2a_{rms}} \qquad (5)$$

where a^+ and a^- are the positive and negative amplitudes, respectively, exceeded for a fraction of time specified by P(a). These amplitudes are determined from the cumulative amplitude probability density P(a):

$$\text{PROB}[a^- \le a(t) \le a^+] = \int_{a-}^{a+} p(a)da = P(a^+) - P(a^-) \qquad (6)$$

where $P(a^+)$ and $P(a^-)$ define the required probability in the cumulative probability distribution function, i.e.

$$P(a^+) - P(a^-) = P(a) \qquad (7)$$

Impulsiveness does not depend on the amplitude distribution possessing a particular shape. It will, therefore, apply equally to Gaussian and non-Gaussian signals. Furthermore, it is evident that the impulsiveness equals the crest-factor when $a^+=a_{max}$ and $a^-=a_{min}$.

Several investigators have explored time domain methods. Zseleczky(1989) and Molyneaux and Roddan (1991) have looked at the distribution of peak accelerations in high-speed planing-boats. Various statistics can be derived from these distributions, such as the average peak and average of the one/tenth highest peaks to characterize the severity of the ride experienced in these craft. The method of determination of the peaks is not a trivial matter as Zseleczky points out (1989). Careful attention must be paid to the criteria by which peaks are defined and recognized.

Dose estimates

A general expression for the integrated effect of exposure to vibration for an extended period of time may readily be formed from a single-number measure of the magnitude of the hazard $a_w(t)$ at time T, by the dose $D(a_w,T)$, where:

$$D(a_w,T)=\{\int_0^T [a_w(t)]^m dt\}^{1/r} \qquad (8)$$

where $a_w(t)$ = weighted acceleration weighted acceleration

To establish an appropriate measure of the dose using equation (8), it will be necessary to determine, as a function of time, an appropriate measure of:

1. The magnitude of the stimulus

2. The relative hazard presented by the vibration at different frequencies

3. The combined effect of the vibration in different directions

In addition, it will be necessary to specify values for parameters m and r. Several papers, (Wikstrom, Kjellberg, and Dallner, 1991; Griffin, 1987; Griffin, 1984), and the British Standard 6841 (British Standards Institution, 1987) have suggested dose measures for characterizing human response to vibration. Two commonly used dose measures are the rms dose (when m=2 and r=2), and the rmq dose or VDV (when m=4 and r=4). The latter is the basis of the British standard 6841 (British Standards Institution, 1987).

Wikstrom, Kjellberg and Dallner (1991) compared different methods for assessing the effect of mechanical shocks on discomfort. This study considered peak-value measures, higher order time-mean measures (to root-mean-ten), dose measures and various biodynamic model response measures. The study showed that a dose calculation based on rms or rmq , but with r=1 in equation (8), gave marginally better predictions of discomfort from shocks.

Material fatigue analysis

Recent papers,(Carter et al. 1981; Lafferty, 1978) have suggested that tissue and bone exhibits fatigue (i.e. a reduction in the ability to carry a load) in a manner that follows similar relationships to those for material fatigue in metals. Although the microscopic details of failure differ substantially between living and inert materials, the principles governing cumulative fatigue in metals may be applied to living tissue. These principles were first developed by Miner (1945). Simply put, if

the number of cycles, N_i, to a given stress, S_i corresponds to a failure criterion, then a lower number of cycles n_i to the same stress level, S_i, produces a partial fatigue proportional to n_i/N_i. If successive loadings occur at various stress levels such that there are n_1 loadings to S_1, n_2 loadings to S_2 etc., then the cumulative fatigue is:

$$c = \sum_{i=1}^{m} n_i/N_i \qquad (9)$$

where:

n_i = number of cycles to the i-th stress level

N_i = number of cycles to failure at the i-th stress level

m = number of stress levels

A significant feature of cumulative fatigue concept is the hypothesis that failure occurs when $c = 1.0$. Values of $c < 1.0$ indicate the fraction of fatigue life which has been consumed.

Applying this formula, together with knowledge of the material properties of parts of the body such as inter-vertebral discs, or viscera and loading information from sophisticated biodynamic models, an estimate of the number of cycles to failure can be made. The use of this approach to predict the development of back injury has been explored by Sandover (1985) (See section on Biomechanics).

Dowling (1972), reports methods of evaluating complicated loading histories of engineering materials or metals. With complicated load histories, Dowling points out that the definition of a load cycle is important and demonstrates six methods for counting the cycles. Such complicated load histories may be present in the various structures within the human body when subject to a vibration exposure. If fatigue criteria are to be applied, the cycle counting methods reported by Dowling may be appropriate.

Techniques for Separating Impacts from Continuous, Random Vibrations

Amplitude distribution methods

The separation of impacts from random vibration can be performed by amplitude windowing. In this method, vibration amplitudes exceeding a predetermined threshold are considered to define an impact which may then be characterized by peak

amplitude and time interval between occurrences. This method of peak analysis is most suited to signals in which the threshold (or thresholds) can be related to the minimum amplitude(s) associated with human response(s).

Although not reported in the literature, the instantaneous calculation of ratios of higher order mean values to rms values (Table 1) will immediately establish the presence of an impact within a background of random vibration, irrespective of the magnitude of the signals.

Auto-regression techniques

Auto-regression (AR), which is essentially a time domain method (Bagshaw and Johnson, 1977; Hollkamp and Batill, 1990; Haggan and Ozaki, 1980; Basseville and Benveniste, 1983), formulates a mathematical model of a signal or process based on its recent past values. Arakawa et al (1986; 1986) report a technique of separating stationary and non-stationary signal components using AR methods. The authors used AR techniques to formulate a model of an EEG wave form. When the electro-encephalogram (EEG) signal deviates significantly from this model a spike in the wave form is thus detected and then separated out. Application of this procedure to the analysis of vibration data containing non-stationary impacts is likely to be useful. Analysis of the random component of the signal could proceed using standard frequency domain techniques, while evaluation of the separated impacts could be performed using peak analysis, or some other method.

Adaptive filtering techniques

A time domain technique called adaptive filtering (AF) is a signal processing method for removing noise and unwanted artifacts from a signal record (Jewell and Jones, 1983; Kao et al. 1989; Liu et al. 1990; Gritsyk and Palenichka, 1989; Iyer, Ploysongsang, and Ramamoorthy, 1990; Widrow et al. 1975; Thakor and Zhu, 1991; Wong and Kwong, 1991).

The basic technique behind AF is the processing of two signal inputs; one containing the desired signal plus noise, and another (reference) input containing noise, which is correlated with the noise of the primary input. The combined signals, primary plus reference, are fed to an adaptive filter network, which adjusts its filter coefficients to minimize the total power output of the combined signal. The net effect of this adaptive procedure is to cancel the effect of the noise (or whatever signal is contained in the reference). The attraction of this procedure is that no prior knowledge of the noise spectrum is required as is the case with passive filter networks.

Applications of this technique include adaptive noise canceling, (i.e. power line interference canceling) and separating of the fetal electrocardiogram (ECG) from the mother's ECG, by canceling the latter. Of particular interest to this study is its application of canceling noise and motion artifacts in ECG, EMG, and electrogastrogram (EGG) signals.

Summary

Time domain techniques, combined with appropriate frequency weightings and temporal assessments of the vibration exposure, appear to be the most promising in developing a suitable characterization of vibration signals containing mechanical shocks and repeated impacts. Conventional frequency domain analyses are important for validation and for providing a reference when comparing to existing data.

For example, auto-regressive methods could be used to separate the impulsive, non-stationary components from the stationary continuous background vibration. Analysis of the stationary component could proceed using standard frequency domain techniques while the impulsive non-stationary component would be dealt with using time domain methods. Magnitude estimates would be obtained for the two signal components which could be treated in a manner analogous to complex numbers. In other words, the signal would be characterized by a complex number, a + ib, where, a, is the magnitude estimate of the stationary component and b, is the magnitude estimate of the impulsive component.

Our future work will consist of first choosing a suitable model with which to appropriately weight the frequency components of the input acceleration applied to the human body, ideally in all three x, y, and z axes, with respect to their health effects. A detailed analysis of the amplitude characteristics of the resulting processed signal will be carried out. A collection of methods will be tested including, higher order means, dose measures, peak analysis and their resultant distributions, and auto-regression techniques. Also included in the analysis will be cumulative damage models, at least one of which will include allowance for biological recovery. The most promising of the measures so far obtained will be correlated with the physiological results of the pilot study, to determine which appears most suited to predict the long term effects of mechanical shock and repeated impact on health.

Conclusions

Acute and Chronic Health Disorders

1. Epidemiological studies of whole-body vibration have investigated both subjective symptoms and objective measurements of health disorders. Unfortunately, most studies are poorly designed. Thus, determining well-supported cause-effect relationships is difficult.

2. Evidence exists that long-term exposure to vibration can be harmful to the spine, and possibly gastro-intestinal and cardiovascular systems. Vibration, rather than causing specific pathologies, seems to accelerate the onset of currently recognizable syndromes.

3. There appears to be distinct differences in the mechanisms of acute and chronic injury to the spine caused by exposure to impact and vibration. Severe impact causes fracture of the vertebrae, most commonly in the lower thoracic and upper lumbar region. Chronic back problems are usually in the lumbar region. Vibration-induced nutritional and fatigue mechanisms have been hypothesized.

Vibration Measurement and Standards

4. A large number of studies have involved subjective response to vibration. Much of this data has been used in formulating the International Orgnaization for Standardization (ISO) guidelines for human response to whole-body vibration.

5. Levels of vibration and impact have been collected from a wide range of studies. Some of the highest levels of vertical acceleration and crest-factors were reported for military vehicles. When evaluated using the ISO (2631, 1982) guidelines, some of these vehicles exceeded in minutes the recommended exposure limits.

6. Recent Draft Revisions of ISO 2631, and the British Standard (6841, 1987) incorporate rmq and vibration dose values as the preferred method of characterizing vibration with high crest-factors. Little evidence exists, however, to support use of these standards for signals with shock and repeated impacts of high magnitude. Exposure limits are not contained in the body of the Standards.

7. Two guidelines exist for exposure to repeated impacts, both using the dynamic response index (model predicting spinal injury) as their basis (Air Standardization Coordinating

Committee, 1982; Kanda, 1982). The DRI is limited to impact in the vertical axis, with no consideration of recovery. The predictive power of the DRI has been questioned, and there is no independent validation of Kanda's guideline.

8. In the few investigations that involved subjective response to shock or repeated inpact, the rms and rmq measurements seemed equally good predictors of discomfort; the exponent of best fit depending upon the frequency, separation and anticipation of the shocks, background vibration levels, and prior training.

9. A number of signal processing techniques have been used in other applications which may prove useful in characterizing impulsive signals from TGVs. These include SIWEH, shock spectrum, peak processing and auto-regression techniques.

Human Response to Vibration and Impacts

10. Many of the physiological and biochemical responses to whole-body vibration mimic those produced by exercise, including general stress, muscle fatigue and tissue or organ damage. Few studies in humans have investigated these responses in conditions of repeated impact, and none have investigated the body's ability to recover. Similarly, theories of fatigue failure do not consider tissue recovery through on-going nutritional mechanisms.

11. Measurements of impedance and apparent mass provide useful indications of the response of the body to WBV and impact. However, these measurements do not provide sufficient detail to determine the behaviour of, or stresses acting on, individual systems such as the abdomen and spine.

12. Biodynamic models range from single degree of freedom to three-dimensional and discrete parameter models. Sophisticated models suffer from lack of experimental data to validate their predictions. Most models are not designed to predict chronic health problems. Despite these criticisms, certain biodynamic models have direct relevance to development of a health hazard assessment.

Recommendations for the Health Hazard Assessment

1. Chronic health effects from epidemiological literature suggest that the spine and gastro-intestinal systems are most affected by WBV and repeated impact. Measurements of stress in these systems during simulated TGV exposures can be compared with "safe" stress induced in more familiar environments (walking or light physical work) to assess potential for accelerated onset of chronic health problems.

2. Physiological, biochemical and biomechanical data will be used to indicate deviation from normal responses, to seek correlations with individual and repeated impacts, and to measure recovery following impacts.

3. Physiological stress may be evaluated with ECG spectral components, responsiveness of ECG parameters to repeated impacts and biochemical markers such as LDH and CPK activity. EMG measurements and biochemical markers such as serum electrolytes can indicate muscle fatigue. Tissue damage can be investigated by measurements such as von Willebrand's factor.

4. Correlations between physiological, biochemical and biomechanical changes in response to impact will be used to identify possible chronic health problems.

5. Measurements of spinal acceleration, abdominal displacement and muscle tension (EMG) can be used in biomechanical modeling to calculate internal tissue stresses in response to impact. It may be posible to devise a unified theory of injury which will encompass the mechanisms of both acute damage and chronic degenerative failure.

6. Since available standards and guidelines do not accommodate the high crest-factors and acceleration levels measured in TGVs, there is an obvious requirement for signal characterizations capable of encompasing both impulsive accelerations and multi-axial random vibration.

7. Time domain techniques, combined with appropriate frequency weightings and temporal assessments of the vibration exposure, appear to be the most promising means of characterizing signals containing shocks and repeated impacts. Methods which merit consideration include higher order means, dose measures, peak processing methods, auto-regression and cumulative damage models. Conventional frequency domain analyses are important for validation and for providing a reference when comparing to existing data.

8. Although a subjective response model may be acceptable for exposures to random vibration, it becomes difficult to apply with confidence to shock and repeated impact. In these

circumstances, a biodynamic model may provide a more versatile prediction of human response. Advances in modeling offer the potential of an index incorporating spinal loading, tissue stresses, and fatigue and recovery characteristics of various systems of the body.

9. Epidemiological data of health disorders, combined with vibration levels of various equipment, may help to construct dose-response relationships that can assist in validating the eventual health hazard assessment index.

Measurement Indices for Pilot Test by Priority

- triaxial seatpan accelerations
- EMG of paraspinal muscles
- biochemical markers: LDH, CPK activity, von Willebrand's factor, hemotological factors, serum electrolyte
- ECG: spectral and temporal components
- triaxial spinal accelerations
- gastro-intestinal pressure or displacement
- shoulder/head acceleration
- gastro-intestinal motility
- peripheral blood flow
- subjective assessment
- TTS (possibly)

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Appendix A: Topics Covered by Various Team Members

Topic Area	Primary Team Member
Health effects of vibration and impact from epidemiological research	Judy Village
Subjective response to vibration and repeated impact	Judy Village
Physiological effects of vibration and repeated impact	Julia Rylands
Biochemical effects of vibration and repeated impact	Barb Cameron
Muscle response to vibration and repeated impact	Dan Robinson
Biodynamic response, biomechanics and biodynamic models	Jim Morrison
Standards and guidelines for shock and vibration	George Roddan and Judy Village
Vibration data collected in the field	Judy Village
Signal processing	Malcolm Smith, Tony Brammer and George Roddan

Appendix B
Table B-1

BACK PAIN IN HEAVY EQUIPMENT OPERATORS AND MISCELLANEOUS

	No in Vib. gp	No in Ctrl. gp	Field Data	Vehicle type	General back pain - Ctrl	General back pain - Vib	Low back pain - Ctrl	Low back pain - Vib	Middle back pain - Ctrl	Middle back pain - Vib	Upper back/ shoulder pain - Vib	Neck pain - Vib	Neck pain - Ctrl	COMMENTS
Konda et al (1985)	540			Container Tractor		41.5		60.2			44.6	33.7		
Burdorf and Zondervan (1990)	33	30	1.5-8 Hz	Crane	26.7	60.6	26.7	60.6						
Dupuis and Zerlett (1987)	352	315	0.2-2.2 m/s^2	Earth Moving	54	70	41.6	68.7	6.8	16.5		27	18.2	
Kunz and Meyer (1969)X	52			Earth Moving		33		33						
Hilfert et al (1981)X	352	315		Earth Moving		70								
Rilhimaki et al (1988)	852	674		Earth Moving	75	90	7	27						
Boshuizen et al (1990)	233	Yes		Forklift				p<0.05						prevalence LBP increased with dose->0.25 $yr\ m^2/s^4$ and 10-20% increase LBP
Beevis and Forshaw (1985)	20		0.1 m/s^2	FV/Centurion		55								
Beevis and Forshaw (1985)	28		1.1 m/s^2	FV/M113		35.7		35.7						
Beevis and Forshaw (1985)	18		1.1 m/s^2	FV/Pool/M113		89								significantly greater back pain than Centrulon or Pooldrivers who drive fewer hours
Beevis and Forshaw (1985)	24		1.1 m/s^2	FV/RCR/M113		46								
Rehm and Wieth (1984)	235	314		Heavy Engine	70.1	91.9								
Wilcox et al (1988)	20		0.3-0.65 m/s^2	Log Stacker		70		70						

x obtained from Hulshof and vanZanten, 1987

Note: bolded numbers are statistically significant

Appendix B
Table B-2

BACK PAIN IN TRACTOR DRIVERS

	No in Vib. gp	No in Ctrl. gp	Field Data	Vehicle type	General back pain - Ctrl	General back pain - Vib	Low back pain - Ctrl	Low back pain - Vib	Middle back pain subj - Ctrl	Neck pain - Vib	COMMENTS
Christ and Dupuis (1968)X	137			Tractor		47		47			increasing stomach problems with years of driving
Dupuis and Christ (1972)X	106			Tractor		57		57			
Kohl (1975)X	582			Tractor		61		61			
Rosegger and Rosegger (1960)	371		0.6-1.1 m/s^2	Tractor		30		30			
Seidel and Troster (1970)X	60			Tractor		41		41			
Zimmerman (1966)+	137			Tractor		41					
Paulson (1949)	23			Tractor		43.5				13	
Boshuizen (1990)	450	110	0.6-1.1 m/s^2	Tractor	27.3	38.4	19.1	31.3			
Boshuizen et al (1990)	455	Yes		Tractor				p<0.05			prevalence LBP increased with dose

x obtained from Hulshof & VanZanten, 1987

+ obtained from Dupuis & Zerlett, 1986

Note: bolded numbers are statistically significant

Appendix B
Table B-3

BACK PAIN IN TRUCK, BUS AND CAR DRIVERS

	No in Vib. gp	No in Ctrl. gp	Field Data	Vehicle Type	General back pain - Ctrl	General back pain - Vib	Low back pain - Vib	Upper back/ shoulder pain - Vib	Neck pain - Vib	COMMENTS
Backman (1983)	633			Bus/Truck		40				
Bachman (1983)	1156			Bus/Truck		40			70	pain in shoulders and neck
Cavigneaux and Laffont (1969)X	93			Car/Taxi		8	8			low back pain at moment of investigation
Kelsey & Hardy (1975)	223	711		Misc. Vehicle Drivers						driving 2.75X risk, truck drivers 4.67X risk
Kriston et al (1981)+	94			Truck		72				
Konda et al (1985)	103			Truck		40.8	51.5	38.8	22.3	
Schmidt (1969)+	117			Truck						
Wilcox et al (1988)	13		0.3 - 0.65 m / s^2	Truck		92	92			
Gruber (1976)	3205	1137	0.5 - 1.5 m/ s^2	Truck						
Rehm and Wieth (1984)	54			Truck/Car	70	87				
Rehm and Wieth (1984)	52			Truck/Retired Drivers	70	84.6				
Sakuma (1980)	60			Trucks/Dump		30				pain increases beyond 30-45 hours/month
Fishbein & Salter (1950)	378			Trucks/Tractors						62% doctors replied they'd seen patients with spine disorders related to driving

x obtained from Hulshof & vanZanten, 1987

+ obtained from Dupuis & Zerlett, 1986

Appendix B
Table B-4

BACK AND NECK INJURIES

	No in Vib. gp	No in Ctrl. gp	Vehicle type	Neck Injury - Ctrl	Neck Injury - Vib.	Back Injury - Ctrl	Back Injury - Vib.	COMMENTS
Boshulzen et al (1990)	341	Yes	Crane/Shipyard				p>0.05	not significant but sick leave due to back disorders lasted longer
Boshulzen et al (1990)	743	Yes	Crane/SteelCo.				p<0.05	>28 days back disability-no information about control
Desrosiers et al (1988)	584	2,071	Mining/LHD	3.9	10.5	10.5	19	3 control groups - used office workers for comparison
Boshuizen et al (1990)	423	Yes	Tractor				p>0.05	not significant but higher incidence of first sick leave due to back problems
Boshulzen et al (1990)	689	109	Tractors/Heavy equipment				p<0.05	incidence lost time sick leave due to back disorders significantly higher, relative risk IV disc disease higher and > risk
Hannunkarl (1979)	4347	2799	Train				p<0.05	greater relative risk of disability for engineers than clerks but not train men

Note: bolded numbers are statistically significant

Appendix B
Table B-5

RADIOLOGICAL BACK DISORDERS IN HEAVY EQUIPMENT OPERATORS AND MISCELLANEOUS

	No In Vib. gp	No In Ctrl. gp	FIELD DATA	Vehicle type	Radiological disorders - Ctrl	Radiological disorders - Vib.	IV disc hernia/ rupture - Vib.	Thoracic deg.- Vib.	Lumbar deg. - Ctrl	Lumbar deg.- Vib.	Sciatica - Ctrl	Sciatica - Vib.	Spondylolisthesis - Vib.	Scoliosis - Ctrl	Scoliosis - Vib.	COMMENTS
Beevis and Forshaw (1985)	28		1.1 m/s^2, CF 13	FV/M113		32	7.1									
Burdorf and Zondervan (1990)	33	30	1.5-8 Hz	Crane	10	27					10	27				
Dupuis and Zerlett (1987)	352	315	0.2-2.2 m/s^2	Earth Moving		p<0.05				p<0.05						
Kohne et al (1982)+	352			Excavator		81										
Kunz and Meyer (1969)X	52			Earth Moving		66										
Grigoryan (1989)	88	Yes		Excavator	10	46.5										
Rehm and Wieth (1984)	235	314		Heavy Engine	62.43	80.33										
Spear et al (1976)	667	707		Heavy Equipt.	8.5	8.4										elevated relative risk for 3 diseases
Wilcox et al (1988)	20		0.3 - 0.65 m/s^2	Log Stacker		10	10									
Kersten (1966)+	220			Ship		87										
Louyot et al (1954)+	78			Train		80										
Arnautova-Bulat (1979)	76	45		Train		62										
Bongers et al (1988)	743	662	0.25-0.67 m/s^2	Crane		p<0. 05										long term disability,crane operators 4X IV disc disease and 1.5X higher with 10 yrs
Bongers et al (1988)	743	662	0.25-0.65 m/s^2	Crane		p<0.05										sickness of >28 days - 2X incidence of disc disorders
Dupuis & Zerlett (1987)	251	314	0.3-2.2 m/s^2	Earth Moving	8.6	23.2			53	81				11	10	
Kunz & Meyer (1969)+	49			Earth Moving		50										
Spear et al (1977)	89	95		Heavy Equipt.		p<0.05										slight increase in selection out due to musculo-skeletal disease
Hilfert et al (1981)X	352	315		Earthmoving	53	81			53	81						
Grzesik (1980)	492	Yes	< & > 0.3 m/s^2	Mine Equipt.		p<0.05										higher incidence of problems following vibration exposure
Grzesik (1980)	652	Yes	< & > 0.3 m/s^2	Mine Equipt.		p<0.05										
Mustajoki et al (1978)	50	50		Parachutists				46	36	44						parachutist had greater thoracic & cervical problems, but not lumbar
Kanda (1982)	487	1580	DRI permiss.<1 hr.	Ships/high speed	65	76										
Arnautova-Bulat (1979)	76	45		Train	29	59			49	41			25			

x obtained from Hulshof & vanZanten, 1987
+ obtained from Dupuis & Zerlett, 1987
Note: bolded numbers are statistically significant

Appendix B
Table B-6

RADIOLOGICAL BACK DISORDERS IN TRACTOR DRIVERS

	No in Vib. gp	*Field Data*	*Vehicle type*	*Radiological disorders - Vib.*	*Narrowing IVdisc - Vib.*	*Deformity of spine - Vib.*
Christ and Dupuis (1968)X	137		Tractor	58		
Dupuis and Christ (1972)X	106		Tractor	80		
Kohl (1975)X	582		Tractor	31	31	
Kubic (1966)+	400		Tractor	68		
Lavault (1962)+	107		Tractor	33		
Rosegger and Rosegger (1960)	371	1.5 – 3.6m/s^2	Tractor	70		
Schulze and Polster (1979)+	103		Tractor	80		
Seidel and Troster (1970)X	60		Tractor	28		28
Zimmerman (1966)+	137		Tractor	80		
Christ (1968)+	211		Tractor	50		
Dupuis & Christ (1961)+	137		Tractor	69		

x obtained from Hulshof & vanZanten, 1987

+ obtained from Dupuis & Zerlett, 1986

Appendix B
Table B-7

RADIOLOGICAL BACK DISORDERS IN TRUCK, BUS AND CAR DRIVERS

	No in Vib. gp	No in Ctrl. gp	Field Data	Radiological disorders - Vib.	Radiological disorders - Ctrl	Vehicle type	IV disc hernia/ rupture - Vib.	Thoracic deg.- Vib.	Lumbar deg.- Vib.	COMMENTS
Barbaso (1958)+	213			44		Bus				
Garbe (1981)+	811			50		Bus		10	40	
Gruber and Ziperman (1974)	1448	3542	0.3–3.0m/s^2	p<0.05		Bus				significant difference increased with exposure 16-47%
Cavigneaux and Laffont (1969)X	93			65		Car/Taxi				low back pain at moment of investigation
Kristen et al (1981)+	94			83		Truck				
Schmidt (1969)+	117			79.5	61.1	Truck				
Wilcox et al (1988)	13		0.3–0.65m/s^2	15		Truck	15			
Rehm and Wieth (1984)	54			76.6		Truck/Car				
Rehm and Wieth (1984)	52			65		Truck/Retired Drivers				

x obtained from Hulahod & vanZanten, 1987
+ obtained from Dupuis & Zerlett, 1986
Note: bolded numbers are statistically significant

Appendix B
Table B-8

BACK PAIN IN AIR TRANSPORT OPERATORS

	No in Vib. gp	No in Ctrl. gp	Field Data	Vehicle type	General back pain - Ctrl	General back pain - Vib	Low back pain - Ctrl	Low back pain - Vib	Middle back pain - Ctrl	Middle back pain - Vib	Upper back/ shoulder pain - Vib	Neck pain - Vib	Neck pain - Ctrl	COMMENTS
Froom et al (1986)	373			Air/fighter		23		23						
Braithwaite and Vyrnwy-Jones (1985)*	119		1.0 m/s^2	Air/Helicopter		82		61.3		28.6	7.6	0.8		
Fischer et al (1980)X	221	Yes	1.0 m/s^2	Air/Helicopter		76.5		82.7		1.8	15.5			
Froom et al (1984)X	153	500		Air/Helicopter		15								
Froom et al (1986)	264			Air/Helicopter		57.6		57.6						
Froom et al (1987)	18		1.0 m/s^2	Air/Helicopter/AH-1S		77.8		77.8						helio significantly different from fighter and transport
Shanahan et al (1985)*	802		1.0 m/s^2	Air/Helicopter	30	72.8		73		6	4	3		
Shulte-Winthrop & Knoche (1978)*	145		1.0 m/s^2	Air/Helicopter		51		54		34		17		control group comparison from another study
Singh (1983)*	21		1.0 m/s^2	Air/Helicopter		100								
Sliosberg (1962)*	128		1.0 m/s^2	Air/Helicopter		87.5		75		42.2	27.3			
Froom et al (1986)	165			Air/Transport		17.6		17.6						
Bongers et al (1990)	39	228	0.17-0.44 m/s^2	Air/Civilian/Sik S61N/S76A	17	68	10.5	51.9						
Bongers et al (1990)	87	228	0.44-0.67 m/s^2	Air/fighter/Alouette3/Bolkow 105	17	68	10.5	51.9						
Boshuizen et al (1990)	133	Yes		Air/Helicopter				p<0.05						prevalence of LBP increased with dose-dose of 0.25 yr m^2/s^4 no problems
Froom et al (1986)	373			Air/fighter		23.1		23.1						
Fitzgerald & Crotty (1975)*	664	1124		Air/pilots	37	66								fighter pilots had > sciatica than helio and transport
Metget et al (1986)	232	175		Air/Helicopter	31	45	69	49	20	22		41	36	
Burmeister & Thorma (1992)	88			Air/jet and propeller	52									

x obtained from Hulshof & vanZanten, 1987
* obtained from Chapman, 1990
Note: bolded numbres are statistically significant

Appendix B
Table B-9

RADIOLOGICAL BACK DISORDERS IN AIR TRANSPORT OPERATORS

	No in Vib. gp	No in Ctrl. gp	Field Data	Vehicle type	Radiological disorders - Vib.	Radiological disorders - Ctrl	IV disc hernia/ rupture - Vib.	IVdisc hernia/ rupture - Ctrl	Narrowing IVdisc - Vib.	Sciatica - Ctrl	Sciatica - Vib.	Spondylolisthesis - Vib.	COMMENTS
Bongers et al (1990)	39	228	0.17-0.44/0.17-0.36	Air/Civilian/Sik S61N/S76A	4.5	4.4	4.5	4.4		6.1	11.3		
Froom et al (1986)	373		1.0m / s^2	Air/fighter							25.5		fighter pilots had > sciatica than helio and transport
Fischer (1980) + X	143			Air/fighter	47								
Froom et al 1985	59	64		Air/fighter pilots	45	3.1			45				transport pilots were controls
Bongers et al (1990)	87	228	0.44-0.67/0.29-0.49	Air/fighter/Alouette3/Bolkow 105	4.5	4.4	4.5	4.4		6.1	11.3		
Delahaye et al (1971)X	1000			Air/Heliocopter	80								
Fischer et al (1980)X	221	Yes		Air/Heliocopter									
Froom et al (1984)	153	500		Air/Heliocopter	4.5	1						4.5	
Froom et al (1986)	264			Air/Heliocopter	12.9						12.9		helicopter significantly different from fighter and transport
Fischer et al (1980)+	136			Air/Heliocopter	80	59							
Froom et al (1984)	109	500		Air/Transport	0.9							0.9	
Froom et al (1986)	165			Air/Transport	9.6						9.6		

x obtained from Hulshof & VanZanten, 1987

\+ obtained from Dupuis & Zerlett, 1986

Note: bolded numbers are statistically significant

Appendix B
Table B-10

OTHER HEALTH PROBLEMS IN TRACTOR, AIR TRANSPORT, HEAVY EQUIPMENT & MISCELLANEOUS OPERATORS

	No in Vib. gp	No in Ctrl. gp	Field Data	Vehicle type	Gastro-intestinal (Objective) - Vib.	Gastro-intestinal (Objective) - Ctrl	Gastro-intestinal (Subjective) - Vib.	Renal/ urinary / genital - Ctrl	Renal/ urinary/ genital - Vib.	Hyper-tension - Vib.	Cardiac - Ctrl	Cardiac - Vib.	Respi-ratory - Vib.	Circula-tory - Vib.	Hearing Loss - Ctrl	Hearing Loss - Vib.	COMMENTS
Rehm and Wieth (1984)	235	314		Heavy Engine	35.7	26.1		3.5	5.1		48.4	48.5			35.4	50.2	
Spear et al (1976)	667	707		Heavy Equipment				4.8	4.3		3.7	4.9		1.8			elevated risk for 3 diseases -
Rosegger and Rosegger (1960)	371		1.5-3.6 m/s^2	Tractor	76.1		81.8	84.8									no selection factors
Paulson (1949)	23			Tractor	52												
Hannunkari et al (1978)	437		43% >EL	Train	12.4				6.4	17.8		8.2	19			3.9	
Grzesik (1980)	492	Yes	< & > 0.3 m/s^2	Mine Equipment			p<0.05		p<0.05					p<0.05			higher incidence of problems
Grzesik (1980)	652	Yes	< & > 0.3 m/s^2	Mine Equipment			p<0.05		p<0.05					p<0.05			following vibration exposure
Monaenkova et al (1979)	1806			Mine Equipment						8.0-36		4.0-5.0					
Christ and Dupuis (1968)x	137			Tractor			18.25										
Kohl (1975)x	582			Tractor			24										
Kubic (1966)+	400			Tractor			20										
Seidel and Troster (1950)x	60			Tractor			17										
Beljani et al. (1988)	10								50								

x obtained from Hulshof & vanZanten, 1987
+ obtained from Dupuis & Zerlett, 1986

Appendix B
Table B-11

OTHER HEALTH PROBLEMS IN TRUCK, BUS AND CAR DRIVERS

	No in Vib. gp	No in Ctrl. gp	Field Data	Vehicle type	Gastro-intestinal (Objective) - Vib.	Gastro-intestinal (Objective) - Ctrl	Gastro-intestinal (Subjective) - Vib.	Renal/ urinary /genital - Ctrl	Renal/ urinary/ genital - Vib.	Hyper-tension - Vib.	Cardiac - Vib.	Hearing Loss - Ctrl	Hearing Loss - Vib.	COMMENTS
Gruber and Ziporman (1974)	1448	3542	0.3-3.3 m/s^2	Bus	p<0.05				p<0.05		p<0.05			significant difference increases with exposure 16-47%
Garbe(1981)+	811			Bus			41.3							
Backman (1983)	633			Bus/Truck			43			20	2	13		
Bachman (1983)	1156			Bus/Truck			35			20				pain in shoulders and neck
Schmidt(1969)+	117			Truck	17	9								
Gruber (1976)	3205	1137	0.5-1.5 m/s^2	Truck			p<0.05			p<0.05	p<0.05			
Rehm and Wieth (1984)	54			Truck/Car	35.2	26.1			5.6		35.2		37	
Rehm and Wieth (1984)	52			Truck/Retired	42.3	26.1		3.5	13.5		63.5		46.2	
Sakuma (1980)	60			Trucks/Dump			38							pain increases beyond 30-45 hours/month

+ obtained from Dupuis & Zerlett, 1986

Note: bolded numbers are statistically significant

TOLERANCE LEVELS FROM VARIOUS SOURCES				
Author	**Year**	**Source**	**Direction/Frequency**	**Tolerance m.s-2**
Gorrill & Snyder	1957	maximal tolerance	z	4.0-8.0
Parks & Snyder	1961	"intolerable"	z	7.0-15.0
Chaney	1964	"alarming"	z (5-7 Hz)	13.0-14.0
Dieckmann	1958	intolerable	z	4
Janeway	1948	extreme discomfort	z (3 Hz)	3.3
Goldman	1961	average of 7 sources	z (3 Hz)	3.3
Miwa	1968	3 minute tolerance	z (3-5 Hz)	1.8
Miwa	1968	3 minute tolerance	x,y (1-3 Hz)	2.8
Dupuis	1972	short-time tolerance	z (2-5 Hz)	3.8
Dupuis	1972	short-time tolerance	z (6 Hz)	6
Dupuis	1972	short-time tolerance	z (1 Hz)	4.5
Dupuis et al	1972	30 minute tolerance	z (5 Hz)	2
Dupuis et al	1972	30 minute tolerance	z (2 Hz)	4
Parks	1962	alarming	z (5-7 Hz)	6
Parks	1962	alarming	z (2-3 Hz)	9
Forshaw & Ries	1986	short-time tolerance	z (4-8 Hz) short time	15-20
Forshaw & Ries	1986	1 and 3 minute predicted	z (4-8 Hz) 1-3 minute	7.5-12.5
Magid et al	1960	short-time tolerance	z (5 Hz)	16
Magid et al	1960	3 minute tolerance	z (5-7 Hz)	4
Allen		DRI of 9 converted	z (4 Hz)-1 shock	72
Allen		DRI of 9 converted	z (5 Hz)-1 shock	60
Allen		DRI of 9 converted	z (8.4 Hz)-1 shock	41
Allen		DRI of 6.9 converted	z (4 Hz)-10 shocks	52
Allen		DRI of 6.9 converted	z (5 Hz)- 10 shocks	44
Allen		DRI of 6.9 converted	z (8 Hz)- 10 shocks	30
Weiss et al	1963	NASA-MSC & AMRL	z	30-260
Brinkley	1985	aircraft escape	z major injury - 200-2000 ms	228
Brinkley	1985	aircraft escape	z moderate injury	180
Brinkley	1985	aircraft escape	z low injury	152
Brinkley	1985	aircraft escape	x major injury - 7-25 ms	460
Brinkley	1985	aircraft escape	x moderate injury	350
Brinkley	1985	aircraft escape	x low injury	280
Brinkley	1985	aircraft escape	y major injury	220
Brinkley	1985	aircraft escape	y moderate injury	170
Brinkley	1985	aircraft escape	y low injury	140
Ames et al	1947	pilot ejection studies	z axis-17-25 msec	70-250
Nelson et al	1988	free fall lifeboat criteria	z training	90
Nelson et al	1988	free fall lifeboat criteria	z abandoning	130
Nelson et al	1988	free fall lifeboat criteria	emergency evacua	180
Nelson et al	1988	free fall with durations	10 msec	120
Nelson et al	1988	free fall with durations	10-20 msec	100
Nelson et al	1988	free fall with durations	20-50 msec	80
Shanahan et al	1884	helicopter impact	velocity change	3.4-6.1 m/s

Appendix B
Table B-13

The Effect of Vibration Plus Noise on Temporary Threshold Shift

AUTHOR	Number of Subjects	Expt./Session Time Mins	Work Load Watts	Vibration Level m.s2	Vibration Frequency Hz	Noise Level dBA	Noise Frequency kHz	Temp. °C	ΔTTS2 4kHz	ΔTTS2 6kHz	ΔTTS2 8kHz
Manninen (1982)	90	60/20									
& (1984)			2	2.12	5	90	0.2-16.0		-1 [1]	5	
			2	2.12	5	90	0.2-16.0		2 [2]	4.5 [*]	
			2	2.12	5	90	0.2-16.0		3.5 [3]	4	
			2	2.12	2.8-11.2	90	0.2-16.0		4 [*]	6	
			2	2.12	2.8-11.2	90	0.2-16.0		7 [*]	6.5	
			2	2.12	2.8-11.2	90	0.2-16.0		9.5 [*]	6	
			4	2.12	5	90	0.2-16.0		-0.5 [1]	4.5	
			4	2.12	5	90	0.2-16.0		-2.5 [2]	3.5	
			4	2.12	5	90	0.2-16.0		-0.5 [3]	9.5 [*]	
			4	2.12	2.8-11.2	90	0.2-16.0		2.5 [*]	3.5	
			4	2.12	2.8-11.2	90	0.2-16.0		0.5 [*]	3	
			4	2.12	2.8-11.2	90	0.2-16.0		0.5 [*]	5.5	
			8	2.12	5	90	0.2-16.0		-4.5 [1]	-3	
			8	2.12	5	90	0.2-16.0		-4 [2]	-7.5 [*]	
			8	2.12	5	90	0.2-16.0		-3 [3]	-6	
			8	2.12	2.8-11.2	90	0.2-16.0		-2.5	0	
			8	2.12	2.8-11.2	90	0.2-16.0		-5.5 [*]	-4	
			8	2.12	2.8-11.2	90	0.2-16.0		-5	-5.5 [*]	
Manninen (1983)	13	48/16									
				2.12	5	90	2.0-4.0		~0 [1]	~0 [1]	~2 [1]
				2.12	5	90	2.0-4.0		~1 [2]	~1 [2]	~1 [2]
				2.12	5	90	2.0-4.0		~3 [3]	~0 [3]	~1 [3]
				2.12	5	90	4.0-6.0		~5 [1]	~7 [1]	~3 [1]
				2.12	5	90	4.0-6.0		~6 [2]	~3 [2]	~5 [2]
				2.12	5	90	4.0-6.0		~7 [3]	~3 [3]	~2 [3]
				2.12	5	90	4.0-8.0		~3 [1]	~0 [1]	~0 [1]
				2.12	5	90	4.0-8.0		~1 [2]	~1 [2]	~1 [2]
				2.12	5	90	4.0-8.0		~3 [3]	~3 [3]	~1 [3]
				2.12	5	90	6.0-8.0		~1 [1]	~3 [1]	~0 [1]
				2.12	5	90	6.0-8.0		~1 [2]	~2 [2]	~0 [2]
				2.12	5	90	6.0-8.0		~1 [3]	~1 [3]	~0 [3]
				2.12	5	90	0.2-16.0		~7	~3 [1]	~0 [1]
				2.12	5	90	0.2-16.0		~4 [2]	~5 [2]	~3 [2]
				2.12	5	90	0.2-16.0		~6 [*3]	~5 [*3]	~3 [*3]
	14			2.12	5	90	1.0-2.0		~0 [1]	~2 [1]	~0 [1]
				2.12	5	90	1.0-2.0		~1 [2]	~2 [2]	~1 [2]
				2.12	5	90	1.0-2.0		~2 [3]	~3 [3]	~2 [3]
				2.12	5	90	1.0-4.0		~6 [*1]	~6 [*1]	~3 [1]
				2.12	5	90	1.0-4.0		~5 [2]	~5 [2]	~2 [2]
				2.12	5	90	1.0-4.0		~4 [3]	~5 [3]	~1 [3]
				2.12	5	90	1.0-8.0		~6 [*1]	~3 [1]	~3 [1]
				2.12	5	90	1.0-8.0		~5 [2]	~2 [2]	~3 [2]
				2.12	5	90	1.0-8.0		~5 [3]	~5 [3]	~3 [*3]
				2.12	5	90	0.2-16.0		~4 [1]	~1 [1]	~0 [1]
				2.12	5	90	0.2-16.0		~4 [2]	~6 [*2]	~2 [2]
				2.12	5	90	0.2-16.0		~5 [*3]	~3 [3]	~3 [*3]

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Table B-13

The Effect of Vibration Plus Noise on Temporary Threshold Shift

AUTHOR	Number of Subjects	Expt./Session Time Mins	Work Load Watts	Vibration Level m.s2	Vibration Frequency Hz	Noise Level dBA	Noise Frequency kHz	Temp. °C	ΔTTS2 4kHz	ΔTTS2 6kHz	ΔTTS2 8kHz
Manninen (1983)	11			2.12	5	85	0.2-16.0		-4 *1		
(cont)				2.12	5	85	0.2-16.0		-5 *2		
				2.12	5	85	0.2-16.0		-2 *3		
				2.65	10	85	0.2-16.0		-3 1		
				2.65	10	85	0.2-16.0		-3 2		
				2.65	10	85	0.2-16.0		-1 3		
				2.12	5	98	0.2-16.0		-3 *		
				2.65	10	98	0.2-16.0		-4		
Manninen (1984)	10	48/16									
& Ekbolm				2.12	5	90	0.2-16.0		-8 *1	-10 *1	
				2.12	5	90	0.2-16.0		-7 *2	-9 *2	
				2.12	5	90	0.2-16.0		-7 *3	-8 *3	
				2.44	5	90	0.2-16.0		-8 *1	-13 *1	
				2.44	5	90	0.2-16.0		-8 *2	-13 *2	
				2.44	5	90	0.2-16.0		-9 *3	-9 *3	
Manninen (1985)	108	48/16									
				2.12	5	90	0.2-16.0	20	7	3.4	
				2.12	5	90	0.2-16.0	20	1	-5.8	
				2.12	5	90	0.2-16.0	30	1	4.5	
				2.12	5	90	0.2-16.0	30	2	5.8	
				2.12	2.8-11.2	90	0.2-16.0	20	4	-2.1	
				2.12	2.8-11.2	90	0.2-16.0	20	1	-5	
				2.12	2.8-11.2	90	0.2-16.0	30	3	4.5	
				2.12	2.8-11.2	90	0.2-16.0	30	-3	6.6	
Manninen (1986)	7	80/16									
				2.12	4.4-5.6	90	0.2-16.0		-4 1	-7 1	
				2.12	4.4-5.6	90	0.2-16.0		-4 2	-6 2	
				2.12	4.4-5.6	90	0.2-16.0		-4 3	-5 3	
				2.12	4.4-5.6	90	0.2-16.0		-4 4	-10 4	
				2.12	4.4-5.6	90	0.2-16.0		-3 5	-3 5	
				2.12	2.8-5.6	90	0.2-16.0		-2 1	-7 1	
				2.12	2.8-5.6	90	0.2-16.0		-1 2	-7 2	
				2.12	2.8-5.6	90	0.2-16.0		-1 3	-6 3	
				2.12	2.8-5.6	90	0.2-16.0		-1 4	-6 4	
				2.12	2.8-5.6	90	0.2-16.0		-1 5	-1 5	
				2.12	2.8-11.2	90	0.2-16.0		-5 1	-5 1	
				2.12	2.8-11.2	90	0.2-16.0		-5 2	-4 2	
				2.12	2.8-11.2	90	0.2-16.0		-3 3	-2 3	
				2.12	2.8-11.2	90	0.2-16.0		-5 4	-6 4	
				2.12	2.8-11.2	90	0.2-16.0		-6 5	-1 5	
				2.12	1.4-11.2	90	0.2-16.0		-7 1	-9 1	
				2.12	1.4-11.2	90	0.2-16.0		-5 2	-5 2	
				2.12	1.4-11.2	90	0.2-16.0		-2 3	-5 3	
				2.12	1.4-11.2	90	0.2-16.0		-5 4	-7 4	
				2.12	1.4-11.2	90	0.2-16.0		-3 5	-4 5	

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The Effect of Vibration Plus Noise on Temporary Threshold Shift

AUTHOR	Number of Subjects	Expt./Session Time Mins	Work Load Watts	Vibration Level m.s2	Vibration Frequency Hz	Noise Level dBA	Noise Frequency kHz	Temp. °C	ΔTTS2 4kHz	ΔTTS2 6kHz	ΔTTS2 8kHz
Manninen (1986)				2.12	5	90	0.2-16.0		~6 [1]	~4 [1]	
(cont)				2.12	5	90	0.2-16.0		~4 [2]	~2 [2]	
				2.12	5	90	0.2-16.0		~2 [3]	~3 [3]	
				2.12	5	90	0.2-16.0		~3 [4]	~5 [4]	
				2.12	5	90	0.2-16.0		~5 [5]	~1 [5]	
Manninen (1988)*	60	192/16									
				2.12	5	90	0.2-16.0	35	4 [1]	11 [1]	
				2.12	5	90	0.2-16.0	35	5 [2]	13.5 [2]	
				2.12	5	90	0.2-16.0	35	5.5 [3]	13 [3]	
				2.12	5	90	0.2-16.0	35	6.5 [4]	16 [4]	
				2.12	5	90	0.2-16.0	20	13.5 [1]	11.5 [1]	
				2.12	5	90	0.2-16.0	20	11.5 [2]	12.5 [2]	
				2.12	5	90	0.2-16.0	20	8 [3]	8 [3]	
				2.12	5	90	0.2-16.0	20	8 [4]	8 [4]	
				2.12	2.8-11.2	90	0.2-16.0	35	4.5 [1]	11.5 [1]	
				2.12	2.8-11.2	90	0.2-16.0	35	4.5 [2]	12.5 [2]	
				2.12	2.8-11.2	90	0.2-16.0	35	6 [3]	15 [3]	
				2.12	2.8-11.2	90	0.2-16.0	35	6.5 [4]	13.5 [4]	
				2.12	2.8-11.2	90	0.2-16.0	20	6.5 [1]	7 [1]	
				2.12	2.8-11.2	90	0.2-16.0	20	6.5 [2]	6 [2]	
				2.12	2.8-11.2	90	0.2-16.0	20	7.5 [3]	7 [3]	
				2.12	2.8-11.2	90	0.2-16.0	20	8 [4]	1 [4]	

~ = Read off graph
1 = 1st exposure session
2 = 2nd exposure session
3 = 3rd exposure session
4 = 4th exposure session
5 = 5th exposure session
* = Significant at P <0.05 compared to noise alone
ΔTTS_2 = difference between noise alone and noise & vibration

Appendix B
Table B-14

VIBRATION LEVELS: ON-ROAD WHEELED

Author/Year	Vehicle	Range (m/s^2) az min	az max	az mean	Range (m/s^2) ax min	ax max	ax mean	Range (m/s^2) ay min	ay max	ay mean	Crest Factor-z	Vector Sum (m/s^2)	vdv-z	vdv-x	vdv-y	K-val	Freq-z (Hz)	Freq-x (Hz)	Freq-y (Hz)	Comments
Dupuis (1980)	Ambulance	0.5	1.6																	a z and a y estimated from graph
Griffin (1978)	Bus	0.5	1.75		0.02	0.12		0.05	0.45								6.0-12	1.0-3	1.0-3	a z, y and x estimated from graph
Dupuis (1980)	Bus	0.3	0.7																	a z estimated from graph
Dupuis & Zerlett (1986-7)	Bus	0.4	0.8													8.0-16				
Nelson & Lewis (1989)	Car	0.32	0.98		0.15	0.39		0.12	0.35			0.4-1.1	1.2-3.8	0.57-1.59	0.34-1.57					vector sum = $(ax^2 + ay^2 + az^2)^{1/2}$
Griffin (1978)	Car	0.25	1		0.2	0.15		0.05	0.3								6.0-12	1.0-3	1.0-3	a z, x and y estimated from graph
Dupuis (1980)	Car	0.3	0.7																	a z estimated from graph, rms from dominant third octave
Dupuis (1980)	Car	0.3	0.7																	a z estimated from graph
Dupuis & Zerlett (1986-7)	Car	0.2	0.75													4.0-15.0				
Griffin (1984)	Car/City			0.383			0.112			0.16	4.8	0.43	1.7	0.3	0.5					vector sum = $(ax^2 + ay^2 + az^2)^{1/2}$
Burton & Sandover (1987)	Car/Race			2																
Dupuis (1986)	Car@			2.1																a z measured in bite bar at head
Griffin (1984)	Truck			1.056							3.9		2.9							
Griffin (1978)	Truck	0.7	1.45		0.18	0.45		0.15	0.5								6.0-12	1.0-3	1.0-3	a z, x and y estimated from graph
Boshuizen et al (1990)	Truck											0.78								
Dupuis (1980)	Truck	0.5	1.6																	a z estimated from graph
Wilcox et al (1988)	Trucks			0.73			0.346	0.35	0.58								2.0-8	1.0-4	1.0-4	a z, x and y estimated from allowable exposure in hours
Dupuis & Zerlett (1986-7)	Trucks	0.7	1.4													14-28				
Boulanger et al (1986)	Trucks	0.4	1.25	1	0.15	0.65		0.15	0.65											
Dupuis & Zerlett (1986-7)	Trucks/Road	0.2	0.9													4.0-18				
Griffin (1984)	Van			0.365							<5.0		5							
Griffin (1984)	Van/Country			0.651			0.346			0.503	5.7	0.89	1.8	0.9	1.4					vector sum = $(ax^2 + ay^2 + az^2)^{1/2}$
Boshuizen et al (1990)	Van/Car											0.3-0.4								

@ Acceleration measured at the head

Appendix B
Table B-15

VIBRATION LEVELS: OFF-ROAD VEHICLES

Author/Year	Vehicle	Range (m/s^2) az min	az max	az mean	Range (m/s^2) ax min	ax max	ax mean	Range (m/s^2) ay min	ay max	ay mean	Crest Factor-z	Vector Sum (m/s^2)	vdv-z	vdv-x	vdv-y	K-val	Freq-z (Hz)	Freq-x (Hz)	Freq-y (Hz)	Comments
Dupuis & Zerlett (1986-7)	Crane	0.1	0.8													1.0-16				
Dupuis (1980)	Crawler	0.5	1.6																	
Boshulzen et al (1990)	Dozer											0.6								a z estimated from graph
Dupuis (1980)	Dozer	1.6	2.5																	
Dupuis & Zerlett (1986-7)	Dozers	0.5	2.4																	a z estimated from graph
Dupuis & Zerlett (1986-7)	Excavator	0.3	1.3													12.0-24				
Dupuis (1980)	Excavator	0.3	0.7													6.0-22				
McNulty & Douglas (1985)	Excavator			2.5																a z estimated from graph
Boulanger et al (1978)	Fork-lift			0.8			0.55			0.25							3			a z estimated from graph
Dupuis & Zerlett (1986-7)	Fork-lift	0.4	2.0																	
Dupuis (1980)	Fork-lift	0.5	1.6													8.0-40				
Hansson & Wikstrom (1981)	Forwarder	0.5	1.5		0.1	0.65		0.2	0.75											a z estimated from graph
Crabtree (1982)	FV/AFV	0.1	0.5														1.5-3.0	0.6-1.6	0.6-1.6	a z, x and y estimated from graph
Crabtree (1982)	FV/APC	0.2	0.5																	az estimated from allowable exposure in hours
Crabtree (1982)	FV/AV	0.2	0.5																	az estimated from allowable exposure in hours
Beevis & Forshaw(1985)	FV/Centurion			0.1																az estimated from allowable exposure in hours
Beevis & Forshaw (1985)	FV/M113			1.1																
Dupuis & Zerlett (1986-7)	FV/New	0.45	1.65																	
Dupuis & Zerlett (1986-7)	FV/Old	1.3	4													9.0-33				
Griffin (1986)	FV/Tank			2.5							21		14.5			26-80				
Griffin (1984)	FV/Tank	1.3	2.5									3.3-14.4	3.3-14.5							
Dupuis (1974)	FV/Tank	0.7	9.6		0.08	6.9		0.06	16.9							25-75	1.0-6			
Breen & Calderon (1989)	FV/Tank	0.76	1.07		0.14	0.87		0.13	0.71								1.0-5	1.0-2	1.0-2	
Cole (1977)	FV/Tank	0.35	1.6		0.3	1.5		0.34	1.5		2.0-5					10.0-25	2.0-4	2.0-4	2.0-4	
Dupuis	FV/Tank	4.00	10																	
Bennet et al (1978)	FV/Tank	2.1	3.5								8									a z unweighted
Butler & Madday (1986)	FV/Tank	0.78	1.2		0.16	3		0.16	0.59											
Crabtree (1982)	FV/Tank	0.1	0.5																	a z, x and y estimated from allowable exposure hours
Dupuis & Zerlett (1986-7)	FV/Track	1.5	3.5																	az estimated from allowable exposure in hours
Crabtree (1982)	FV/Tractor	0.3	0.73													30-70				
																				az estimated from allowable exposure in hours

Appendix B
Table B-15 (Continued)

VIBRATION LEVELS: OFF-ROAD VEHICLES

Author/Year	Vehicle	Range (m/s^2) az min	az max	az mean	Range (m/s^2) ax min	ax max	ax mean	Range (m/s^2) ay min	ay max	ay mean	Crest Factor-z	Vector Sum (m/s^2)	vdv-z	vdv-x	vdv-y	K-val	Freq-z (Hz)	Freq-x (Hz)	Freq-y (Hz)	Comments
Crabtree (1982)	FV/Truck	0.3	0.73																	
Dupuis (1980)	Grader	1.6	2.5																	a z estimated from allowable exposure in hours
Dupuis & Zerlett (1986-7)	Grader	0.5	1.6																	a z estimated from graph
Wilcox et al (1988)	Letourneau			0.73			0.346			0.346						12.0-24				
Boulanger et al (1978)	Loader			0.32			0.32			0.32							2.0-4	1.0-4	1.0-4	a z estimated from allowable exposure hours
Dupuis (1980)	Loader	1.6	2.5																	
Howat (1978)	Loaders	0.01	0.7																	a z estimated from graph
Village (1989)	Mine/LHDs	0.71	2.5		0.39	1.5		0.53	0.84											a z rms from dominant third octave
Morrison et al (1991)	Mine/LHDs	0.58	1.23		0.45	1.08		0.45	1.08		20						1.6-5	1.6-3	1.6-3	
Village (1989)	Mine/Trucks	0.2	0.8	0.73	0.1	0.49		0.1	0.37								3.15	1.6-2	1.6-2	
Boshuizen et al (1990)	Shovel											1.1					4.0-10			
Golsse & Hope (1987)	Skidder	0.31	2.19	1.15			0.75			0.82	5-8.79			6.27						
Boileau & Scory (1986)	Skidder			0.83	1.57	2.33				0.85	7.2						2.5			
Boileau (1986-8)	Skidder	0.64	1.1	0.83			0.86			0.96	6.9-9.4	1.99					2.5	1		
Golsse (1989)	Skidder/Track	0.6	1.4		0.5	0.9		0.6	1.55											
Golsse (1989)	Skidder/Wheel	0.6	1.2		0.5	1.25		0.5	1.2											
Dupuis (1980)	Stacker	0.5	1.6																	
Boulanger et al (1978)	Tractor			0.35			0.6			0.6										a z estimated from graph
Monsees et al (1988)	Tractor	0.2	1.8								5-22									
Matthews (1964)	Tractor	2.2	4.5	2.9			0.9			0.9										
Griffin (1984)	Tractor	0.67	2.12		0.59	0.99		0.8	1.86		3-8.5	1.2-2.98	2.9-6.4	2.1-3.2	2.7-4.9					
Boshulzen et al (1990)	Tractor											0.6-1.1								vector sum = $(ax^2 + ay^2 + az^2)^{1/2}$
Dupuis (1980)	Tractor	1.6	2.5																	
Dupuis (1986)	Tractor			2.8																a z estimated from graph
Dupuis & Zerlett (1986-7)	Tractor	0.4	1.25													8.0-25				a z measured in bite bar at head

Appendix B
Table B-16

VIBRATION LEVELS: AIR TRANSPORT

Author/Year	Vehicle	Range az min	Range az max	az mean	Range ax min	Range ax max	ax mean	Range ay min	Range ay max	ay mean	Crest Factor-z	Vector Sum	vdv-z	vdv-x	vdv-y	K-val	Freq-z	Freq-x	Freq-y	Comments
Griffin (1984)	Air/Plane			0.356							4.1		1.4							
Griffin (1990)	Air/Plane	0.36	1.25								4.0-5.3		1.4-4.3							
Griffin (1975)	Helicopter	0.05	1.3		0.05	0.2		0.05	1.3											a z, x and y estimated from graph
Bongers et al (1990)	Helicopter	0.09	1.31		0.04	1.21		0.06	1.31			0.36-0.75					8.0-25	16-25	6.0-20	
Dupuis & Zerlett (1986-7)	Helicopter	0.1	1.5													2.0-31				
Griffin (1987)	Helicopter			0.369			0.096			0.163		1.65								vector sum $=(ax^2+ay^2+az^2)^{1/2}$
Griffin (1984)	Helicopter-Military			1.14			0.31			0.33	3.3	1.23	5.8	0.7	0.8					vector sum $=(ax^2+ay^2+az^2)^{1/2}$
Griffin (1984)	Helicopter-Transport			0.73			0.19			0.19	2.9	0.78	4.7	0.5	0.5					vector sum $=(ax^2+ay^2+az^2)^{1/2}$
Dupuis (1985?)	Helicopter/Ambulance	2.5	4		0.47	1.15		0.7	1.45								5.0-10.0			supine subject, z=ax of helio, x=az of helio, y is same

Appendix B
Table B-17

VIBRATION LEVELS: MISCELLANEOUS VEHICLES

Author/Year	Vehicle	Range (m/s^2) az min	az max	az mean	Range (m/s^2) ax min	ax max	ax mean	Range (m/s^2) ay min	ay max	ay mean	Crest Factor-z	Vector Sum (m/s^2)	vdv-z	vdv-x	vdv-y	K-val	Freq-z (Hz)	Freq-x (Hz)	Freq-y (Hz)	Comments
Nelson & Lewis (1989)	Bicycle	1.36	2.1		0.62	1.2		0.56	0.72			1.6-4.0	5.8-15.8	2.4-4.4	1.98-1.42					
Lewis & Paddan (1990)	Bicycle			3.14																
Dupuis (1986)	Bicycle@			1.6																a z measured in bite bar at head
Dupuis (1986)	Boat@			8.6																a z measured in bite bar at head
Griffin (1984)	Building			0.029							6.1		0.2							
Dupuis (1986)	Horseback@			9.5																a z measured in bite bar at head
Griffin (1984)	Hovercraft			0.473							4.1		2.3							
Griffin (1984)	Hydrofoil			0.078							4.7		0.4							
Nelson & Lewis (1989)	Motorcycle	0.91	1.54		0.2	0.4		0.11	0.23			0.98-1.61	3.48-7.03	0.81-1.66	0.43-0.99					vector sum = $(ax^2 + ay^2 + az^2)^{1/2}$
Irwin (1977)	Parkride	10	40														6.0-8			
Dupuis (1986)	Run@			12.5																a z measured in bite bar at head
Kanda et al (1982)	Ship															0.5-4.9				
Griffin (1984)	Ship			0.48							4.1		0.3							
Dupuis & Zerlett (1986-7)	Ships	0.2	0.7													4.0-14				
Johanning et al (1991)	Subway	0.27	0.69	0.37	0.07	0.17		0.14	0.48			0.55					2.5-12	1.0-2	1.0-2	
Griffin (1978)	Train			0.8			0.05			0.12										a z, x and y estimated from graph
Heino et al (1978)	Train			0.5						0.75							2.0-4.0	2.0-2.5	1.25-4	a z estimated from graph and a y estimated from allowable exposure in hours
Dupuis & Zerlett (1986-7)	Train	0.3	0.6													6.0-12				
Dupuis (1986)	Walk@			1.9-4.0																a z measured in bite bar at head
Barton & Hefner (1976)	Walk@	0.35	0.41														2.0-3			a z measured in bite bar at head

@ Acceleration measured at the head

Appendix C: List of abbreviations and symbols

AF - adaptive filtering

AFV - armoured fighting vehicle

AGARD - Advisory Group for Aerospace Research and Development

ANS - autonomic nervous system

AR - auto-regression

ATP - adenosine triphosphate

ATPase - adenosine triphosphatase

BW - body weight

Ca^{2+} - calcium ion

cAMP - cyclic adenosine monophosphate

cGMP - cyclic guanosine monophosphate

CK - creatine kinase

CKr - creatine kinase released to the circulation

CPK - creatine phosphokinase

CSM - complex spine model

DC - direct current

DRI - dynamic response index

ECG - electrocardiography

EGG - electrogastrography

EL - exposure limit

EMG - electromyography

FDP - fatigue decreased proficiency boundary

FFT - fast Fourier transform

FV - fighting vehicle

g - acceleration due to gravity ($9.81\ m.s^{-2}$)

G1-6-PDH - glucose 1,6 phosphate dehydrogenase

GI - gastro-intestinal

Gz - vertical acceleration

HHA - health hazard assessment

5-HIAA - 5 hydroxyindoleacetic acid

5-HT - 5 hydroxytryptamine (serotonin)

Hz - Hertz

IEMG - integrated electromyography

IMP - inosine monophosphate

IFFT - inverse fast Fourier transform

ISO - International Organization for Standardization

IV - intervertebral

K^+ - potassium ion

kHz - kilohertz

kN - kilonewtons

LDH - lactate dehydrogenase

LHD - load-haul-dump vehicle

MBF - myocardial blood flow

Mg^{2+} - magnesium ion

$m.s^{-2}$ - meters per second squared

ms - miliseconds

MUAP - motor unit action potential

MVC - Maximum voluntary contraction

N - newtons

Na^+ - sodium ion

NIOSH - National Institute for Occupational Safety and Health

PNS - parasympathetic nervous system

PTS - permanent threshold shift

rms - root-mean-square

rmq - root-mean-quad

SDH - succinate dehydrogenase

SIWEH - smoothed instantaneous wave energy history

SLL - spondylolisthesis

SMDS - skeletal muscle damage size

SNS - sympathetic nervous system

SR - sarcoplasmic reticulum

SSM - simplified spine model

TGV - tactical ground vehicles

TTS - temporary threshold shift

USN - United States Navy

vWF - von Willebrand's factor

VWF - vibration white finger

WBV - whole-body vibration